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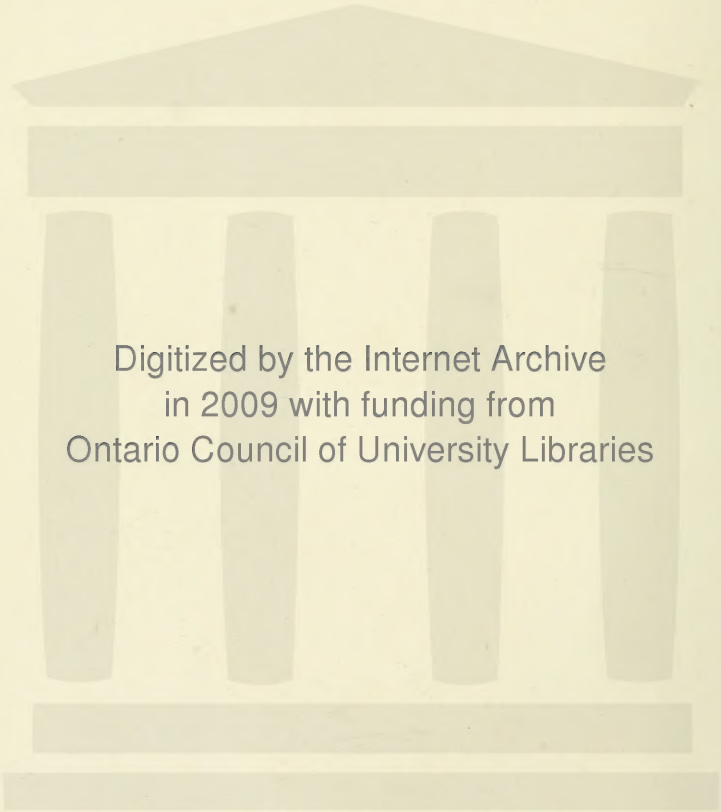
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TRANSACTIONS

OF THE

AMERICAN

SURGICAL ASSOCIATION

VOLUME THE THIRTY-NINTH

EDITED BY

JOHN H. JOPSON, M.D.

RECORDER OF THE ASSOCIATION

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OFFICERS OF THE ASSOCIATION

ELECTED JUNE, 1921

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JOHN M. T. FINNEY, M.D.

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ALEXANDER PRIMROSE, M.D.

JAMES E. THOMPSON, M.D.

SECRETARY

JOHN H. GIBBON, M.D.,
1608 Spruce Street, Philadelphia.

TREASURER

CHARLES H. PECK, M.D.,
30 West Fiftieth Street, New York City.

RECORDER

JOHN H. JOPSON, M.D.,
1824 Pine Street, Philadelphia, Pa.

COUNCIL

LEWIS S. PILCHER, M.D.
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CHARLES H. PECK, M.D.
JOHN H. JOPSON, M.D.

Ex-officio.

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RECORDER, SECRETARY AND TREASURER, *ex-officio.*

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PRESIDENT, SECRETARY, RECORDER AND THE CHAIRMAN OF THE ADJUNCT LOCAL COMMITTEE OF ARRANGEMENTS, *ex-officio.*

COMMITTEE ON ARCHIVES

(Report by Title.)

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ARPAD G. GERSTER, M.D.
CHARLES N. DOWD, M.D.

Place of next meeting:

WASHINGTON, D. C.

ADJUNCT LOCAL COMMITTEE OF ARRANGEMENTS

JAMES F. MITCHELL, M.D.
GEORGE TULLY VAUGHAN, M.D.
RANDOLPH WINSLOW, M.D.

LIST OF THE PRESIDENTS

OF THE

ASSOCIATION FROM THE TIME OF ITS INSTITUTION

ELECTED

1880. ‡SAMUEL DAVID GROSS, M.D., LL.D., D.C.L. (Oxon.), LL.D. (Cantab.), LL.D. (Edin.)—Founder of the Association.
1883. ‡EDWARD MOTT MOORE, M.D., LL.D.
1884. ‡WILLIAM THOMPSON BRIGGS, M.D.
1885. ‡MOSES GUNN, A.M., M.D., LL.D.
1886. ‡HUNTER McGUIRE, M.D., LL.D.
1887. ‡D. HAYES AGNEW, M.D., LL.D.
1888. ‡DAVID WILLIAMS CHEEVER, M.D., LL.D.
1889. ‡DAVID W. YANDELL, M.D.
1890. ‡CLAUDIUS HENRY MASTIN, M.D., LL.D.
1891. ‡PHINEAS SANBORN CONNER, M.D., LL.D.
1892. ‡NICHOLAS SENN, M.D., Ph.D., LL.D.
1893. ‡JAMES EWING MEARS, A.M., M.D., LL.D.
1894. FREDERIC SHEPARD DENNIS, M.D.
1895. ‡LOUIS McLANE TIFFANY, A.M. (Cantab.), M.D.
1896. JOHN COLLINS WARREN, M.D., LL.D. (Jefferson) Hon. F.R.C.S. (Eng.).
1897. ‡THEODORE F. PREWITT, M.D.
1898. WILLIAM W. KEEN, A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. F.A.C.S.
1899. ROBERT F. WEIR, A.M., M.D., Hon. F.R.C.S. (Eng.).
1900. ‡ROSWELL PARK, M.D.
1901. ‡DE FOREST WILLARD, A.M., M.D., Ph.D.
1902. ‡MAURICE H. RICHARDSON, A.B., M.D.
1903. ‡NATHANIEL P. DANDRIDGE, M.D.
1904. ‡GEORGE BEN JOHNSTON, M.D.
1905. ALBERT VANDER VEER, M.D.
1906. ‡DUDLEY P. ALLEN, M.D., LL.D.
1907. WILLIAM H. CARMALT, M.D.
1908. ‡C. B. G. de NANCRÈDE, A.M., M.D., LL.D.

‡ Deceased.

ELECTED

1909. RUDOLPH MATAS, M.D.
1910. RICHARD H. HARTE, M.D.
1911. ARPAD G. GERSTER, M.D., CHIR.D.
1912. CHARLES A. POWERS, A.M., M.D.
1913. WILLIAM J. MAYO, A.M., M.D., D.SC., F.R.C.S. (EDIN.), LL.D. (TOR.).
1914. GEORGE E. ARMSTRONG, C.M., M.D.
1915. ROBERT G. LE CONTE, A.B., M.D.
1916. SAMUEL J. MIXTER, S.B., M.D.
1917. THOMAS W. HUNTINGTON, A.B., M.D., LL.D.
1918. LEWIS S. PILCHER, A.M., M.D., LL.D.
1919. GEORGE E. BREWER, M.D.
1920. JOHN B. ROBERTS, M.D.
1921. JOHN M. T. FINNEY, M.D.

NOTICE

This volume of TRANSACTIONS contains the papers read before the Association at the Meeting held June 14, 15 and 16, 1921.

The Association assumes no responsibility for the statements and opinions published in this volume.

FELLOWS

OF THE

AMERICAN SURGICAL ASSOCIATION

MAY, 1921

* Denotes Original Fellows.
† Denotes Senior Fellows.
‡ Denotes Deceased Fellows.

ELECTED

†1890. Abbe, Robert, M.D., 13 West Fiftieth Street, New York. Senior Surgeon to St. Luke's Hospital. *Vice-President*, 1901. *Council*, 1908.

1902. Andrews, E. Wyllys, A.M., M.D., 122 S. Michigan Avenue, Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Surgeon to St. Luke's Hospital and Michael Reese Hospital; Surgeon-in-Chief to Cook County Hospital; Consulting Surgeon to the Provident Hospital.

1916. Archibald, Edward William, B.A., M.D., C.M., 52 Westmount Boulevard, Montreal, Canada. Lecturer in Clinical Surgery, McGill University; Assistant Surgeon, Royal Victoria Hospital; Associate Surgeon, Children's Memorial Hospital.

1901. Armstrong, George E., M.D., C.M., C.M.G., LL.D. (Queens), D.Sc. (Liverpool), 320 Mountain Street, Montreal. Professor of Surgery and of Clinical Surgery, McGill University; Chief Surgeon to the Royal Victoria Hospital, Consulting Surgeon to the Montreal General Hospital, the Western Hospital and to the Protestant Hospital for the Insane, Verdun; Colonel, C.A.M.C. *President*, 1914.

ELECTED

1913. Ashhurst, Astley Paston Cooper, A.B., M.D., 1620 Spruce Street, Philadelphia. Instructor in Surgery in the University of Pennsylvania; Surgeon to the Episcopal Hospital; Surgeon to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases; Colonel, Medical Reserve Corps, U. S. Army.

1920. Balch, Franklin G., A.M., M.D., F.A.C.S., 279 Clarendon Street, Boston, Mass. Chief of East Surgical Service, Massachusetts General Hospital; Surgeon at the Faulkner Hospital; Associate in Surgery, Graduate Courses in Medicine, Harvard Medical School.

1917. Balfour, Donald Church, M.B., M.D., Rochester, Minn. Associate Professor of Surgery, University of Minnesota; Surgeon to St. Mary's Hospital, Rochester.

1906. Barrow, David, M.D., 148 Market Street, Lexington, Ky. Surgeon to St. Joseph and Good Samaritan Hospitals; recently, Lieut.-Col., M.C.; Commanding Officer, Base Hospital 40.

†1882. Barton, James M., A.M., M.D., 32 North New Hampshire Ave., Atlantic City, N. J.

1915. Besley, Frederic A., M.D., F.A.C.S., 104 South Michigan Avenue, Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Attending Surgeon to the Mercy and Cook County Hospitals.

1900. Bevan, Arthur Dean, M.D., 122 S. Michigan Boulevard, Chicago. Professor and Head of the Department of Surgery, Rush Medical College, University of Chicago; Surgeon to Presbyterian Hospital.

1901. Binnie, J. F., M.A., M.B., C.M. (Aberdeen), 917 Rialto Bldg., Kansas City, Mo. *Vice-President*, 1907 and 1919.

1918. Blair, Vilray Papin, M.D., 4654 Pershing Ave., St. Louis, Mo. Professor of Oral Surgery, Washington University Dental School; Associate in Surgery, Washington University Medical School; Visiting Surgeon, St. Luke's Hospital; Assistant Surgeon in St. Louis Children's Hospital and Barnes Hospital.

1906. Blake, John Bapst, A.M., M.D., 657 Boylston Street, Boston, Mass. Assistant Professor in Surgery, Harvard Medical School; Surgeon-in-Chief to the Boston City Hospital; Consulting Surgeon to Long Island Hospital; Consulting Surgeon to Boston Insane Hospital; Consulting Surgeon to the Reformatory for Women, Framingham, Mass.

ELECTED

1902. Blake, Joseph A., M.D., 116 East 53rd Street, New York, N. Y.

1901. Bloodgood, Joseph Colt, B.S. (Wisc., 1888), M.D. (Penna., 1891); office, 904 N. Charles St., Baltimore, Md.; residence, 18 Midvale Road, Roland Park, Md. Associate Professor of Clinical Surgery, Johns Hopkins University; Surgeon to St. Agnes's Hospital, Baltimore.

1913. Bottomley, John T., A.B., LL.D., M.D., 165 Beacon Street, Boston. Surgeon-in-Chief, Carney Hospital.

†1890. Bradford, Edward Hickling, A.B., M.D., 133 Newbury Street, Boston. Surgeon to the Children's and the Samaritan Hospitals; Assistant Professor of Orthopedic Surgery, Harvard University.

1900. Brewer, George Emerson, M.D., 16 East Sixty-fourth Street, New York City. Emeritus Professor of Surgery in College of Physicians and Surgeons, Columbia University; Consulting Surgeon to the Presbyterian Hospital, Roosevelt, City, Woman's, St. Vincent's, Manhattan Eye, Ear, Nose and Throat Hospitals, and to the Ophthalmic and Aural Institute, New York; to the Muhlenberg Hospital of Plainfield, N. J., the Flushing and Perth Amboy Hospitals. *Vice-President*, 1913. *President*, 1919.

1917. Brewster, George W. W., A.B., M.D., F.A.C.S., 213 Beacon Street, Boston, Mass. Associate in Surgery, Harvard Graduate School; Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon to Frost Hospital, Chelsea, Mass.; Leonard Morse Hospital, Natick, Mass.; Milford Hospital, Milford, Mass.

1915. Brinsmade, W. B., A.B., M.D., 172 Clinton St., Brooklyn, N. Y. Consulting Surgeon to St. Christopher's Hospital; Senior Surgeon, Brooklyn Hospital; Commander, U.S.N.R.F., M.C.

1911. Bruce, Herbert A., M.D. (Toronto Univ.), L.R.C.P. (London), F.R.C.S. (England), 64 Bloor Street East, Toronto, Canada. Associate Professor of Clinical Surgery, University of Toronto; Surgeon to the Toronto General Hospital; Colonel, Canadian Army Medical Corps and Royal Army Medical Corps; Late Consulting Surgeon to the British Armies in France.

1911. Buchanan, J. J., A.M., M.D., Ph.D., 1409 N. Highland Avenue, Pittsburgh, Pa. Emeritus Professor of Surgery, University of Pittsburgh; Surgeon to Mercy Hospital.

ELECTED

1900. Bunts, Frank E., M.D., Euclid Avenue and 93rd Street, Cleveland, Ohio. Professor of Principles of Surgery and Clinical Surgery in Medical College of Western Reserve University; Visiting Surgeon and Chief of Staff to St. Vincent's Charity Hospital.

1921. Campbell, William Francis, A.B., M.D., F.A.C.S., 394 Clinton Avenue, Brooklyn, N. Y. Emeritus Professor of Surgery, Long Island College Hospital; Surgeon-in-Chief to the Trinity Hospital; Consulting Surgeon to the Methodist Episcopal, East New York and Brownsville, Long Island College, Coney Island, Swedish, Jamaica, and Zion Hospitals.

†1885. Carmalt, William H., A.M., M.D., 261 St. Ronan Street, New Haven, Conn. Emeritus Professor of Surgery in Yale University; President and Consulting Surgeon to the New Haven Hospital. *President*, 1907; *Vice-President*, 1895; *Member of Council*, 1908.

1909. Carrel, Alexis, L.B., Sc.B., M.D., Rockefeller Institute for Medical Research, Sixty-sixth Street and Avenue A, New York, N. Y.

†1896. Carson, Norman Bruce, M.D., 303 Humboldt Bldg., St. Louis, Mo. Surgeon to St. Louis, Mullanphy Hospital and to the St. Vincent Insane Asylum, etc. *Vice-President*, 1903.

1920. Clinton, Marshall, M.D., 556 Franklin Street, Buffalo, New York. Associate Professor of Surgery, University of Buffalo, Department of Medicine; Professor of Oral Surgery, University of Buffalo, Dental Department; Attending Surgeon, Buffalo General, Erie County, and Buffalo City Hospitals.

1910. Clopton, Malvern Bryan, M.D., 405 Humboldt Building, St. Louis, Mo. Associate in Surgery, Washington University Medical School; Visiting Surgeon, St. Luke's Hospital; Assistant Surgeon, Barnes Hospital; Associate Surgeon, St. Louis Children's Hospital.

1916. Codman, Ernest Amory, A.B., M.D., 227 Beacon St., Boston, Mass.

1898. Coley, William B., M.D., 6 West Fiftieth Street, New York. Attending Surgeon to the Memorial Hospital; Associate Surgeon to the Hospital for Ruptured and Crippled, New York.

ELECTED

1914. Cotton, Frederick J., A.B., M.D., A.M., 520 Commonwealth Avenue, Boston, Mass. Visiting Surgeon to the Boston City Hospital; Associate in Surgery, Harvard School for Graduate Instruction in Medicine; Consultant in Surgery, United States Public Health Service, Northeastern District; also Consultant in Surgery, New England Hospital for Women and Children.

1905. Crile, George W., M.D., Euclid Avenue and 93rd Street, Cleveland, Ohio. Professor of Surgery, School of Medicine, Western Reserve University; Visiting Surgeon to Lakeside Hospital.

1906. Cushing, Harvey, A.M., M.D., ScD., LL.D., F.R.C.S., The Peter Bent Brigham Hospital, Boston. Professor of Surgery, Harvard University.

†1896. Cushing, Hayward Warren, A.B., M.D., 70 Commonwealth Avenue, Boston, Mass. Consulting Surgeon, Boston City Hospital.

1897. Da Costa, John Chalmers, M.D., 2045 Walnut Street, Philadelphia. Samuel D. Gross Professor of Surgery, Jefferson Medical College; Surgeon to the Hospital of the Jefferson Medical College; Consulting Surgeon to the Philadelphia General Hospital, Saint Joseph's Hospital and Misericordia Hospital.

1917. Darrach, William, A.B., M.A., M.D., 19 East 65th Street, New York City. Associate Professor of Surgery and Dean of the Medical Faculty, College of Physicians and Surgeons, Columbia University; Consulting Surgeon, Memorial Hospital, Morristown, N. J.

1916. Davis, Carl B., A.B., M.D., 122 South Michigan Boulevard, Chicago, Ill. Assistant Professor of Surgery, Rush Medical College; Associate Surgeon, Presbyterian Hospital, Chicago.

1917. Davis, John Staige, Ph.B., M.D., F.A.C.S., Severn Apartments, Baltimore, Md. Instructor in Clinical Surgery, Johns Hopkins University; Visiting and Plastic Surgeon, Union Protestant Infirmary, Hospital for the Women of Maryland, Children's Hospital School; Assistant Visiting Surgeon and Dispensary Surgeon, Johns Hopkins Hospital.

1919. Davis, Lincoln, A.B., M.D., 217 Beacon St., Boston, Mass. Associate in Surgery, Harvard Graduate School of Medicine; Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon, Massachusetts Charitable Eye and Ear

ELECTED

Infirmary, Heywood Memorial Hospital, Gardner, Milford Hospital, Milford.

1912. Deaver, Harry C., M.D., 1701 Spruce Street, Philadelphia. Professor of Surgery, Woman's Medical College of Pennsylvania; Surgeon-in-Chief to Kensington Hospital for Women; Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary Drexel Home.

1892. Deaver, John B., M.D., Sc.D., LL.D., 1634 Walnut Street, Philadelphia. John Rhea Barton Professor of Surgery, University of Pennsylvania, Philadelphia; Surgeon-in-Chief to the Lankenau Hospital, Philadelphia.

1910. Delatour, H. Beeckman, M.D., 73 Eighth Avenue, Brooklyn, N. Y. Attending Surgeon, St. John's and Norwegian Hospitals; Consulting Surgeon, Kingston Avenue Hospital, Brooklyn, St. Mary's Hospital, Jamaica, and Northport Hospital.

†1882. Dennis, Frederic Shepard, M.D., F.R.C.S. (Eng.), 62 East Fifty-fifth Street, New York. Professor Emeritus of Clinical Surgery, Cornell University, New York City; Consulting Surgeon to the Bellevue and Attending Surgeon to St. Vincent's Hospitals; Consulting Surgeon to St. Joseph's Hospital, Yonkers, and Montefiore Home, New York City, Litchfield County Hospital, Winsted, Conn. *President*, 1894; *Vice-President*, 1887; *Member of Council*, 1889.

1910. Dowd, Charles N., A.M., M.D., 127 West Seventy-second Street, New York, N. Y. Professor of Clinical Surgery, Columbia University; Attending Surgeon, Roosevelt Hospital; Consulting Surgeon, Memorial Hospital and St. Mary's Free Hospital for Children, New York.

1914. Downes, William A., M.D., 424 Park Avenue, New York, N. Y. Assistant Professor of Clinical Surgery, Columbia University, College of Physicians and Surgeons; Attending Surgeon, St. Luke's, Memorial and Babies' Hospitals; Consulting Surgeon, Manhattan State Hospital, Hospital for Ruptured and Crippled, Infirmary for Women and Children, New York, United Hospital, Port Chester, N. Y.

1919. Eastman, Joseph Rilus, A.M., B.Sc., M.D., F.A.C.S., 331 N. Delaware St., Indianapolis, Ind. Professor of Surgery, Indiana University School of Medicine; Visiting Surgeon to City Hospital.

ELECTED

1901. Eliot, Ellsworth, Jr., M.D., 34 East Sixty-seventh Street, New York. Surgeon to the Presbyterian Hospital.

†1893. Elliot, John Wheelock, A.M., M.D. (Harvard), 124 Beacon Street, Boston. Surgeon to Massachusetts General Hospital, 1893–1907; Clinical Instructor in Gynecology, 1888–9, and Lecturer on Surgery, 1900–5, Harvard Medical School; Chairman, N. E. Surgical Dressings Committee, 1917.

1911. Elting, Arthur W., A.B., M.D., LL.D., 119 Washington Avenue, Albany, N. Y. Professor of Surgery in the Albany Medical College; Surgeon to the Albany Hospital, and Surgeon to the Child's Hospital, Albany, N. Y.

†1896. Estes, William Lawrence, A.M., M.D., South Bethlehem, Pa. Director and Surgeon-in-Chief of St. Luke's Hospital, Lecturer on Hygiene at Lehigh University.

1898. Eve, Duncan, A.M., M.D., Eve Building, Nashville, Tenn. Professor of Surgery and Clinical Surgery, Medical Department, Vanderbilt University; Chief Surgeon of the N. C. & St. Louis Railway; Consulting Surgeon to Nashville City, St. Thomas and Woman's Hospitals.

1899. Finney, John M. T., M.D. *President.* 1300 Eutaw Place, Baltimore, Md. Associate Professor of Surgery, Johns Hopkins University.

1921. Fisher, William A., Jr., M.D., 715 Park Avenue, Baltimore, Md. Associate in Clinical Surgery, Johns Hopkins University; Assistant Visiting Surgeon, Johns Hopkins Hospital; Visiting Surgeon, Union Memorial Hospital, Church Home and Infirmary, Hospital for the Women of Maryland, Children's Hospital School.

1915. Flint, Joseph Marshall, B.S., M.A., M.D., 320 Temple Street, New Haven, Conn. Professor of Surgery at Yale University.

1904. Frazier, Charles Harrison, M.D., 1724 Spruce Street, Philadelphia, Pa. Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University Hospital.

1898. Freeman, Leonard, B.S., M.D., 424 Metropolitan Building, Denver, Col. Professor of Surgery, Medical Department, University of Colorado; Surgeon to Denver City and County, St. Joseph's, and National Jewish Hospitals; Consulting Surgeon, Children's and Mercy Hospitals, Denver, Col. *Vice-President,* 1908.

1910. Gage, Homer, A.M., M.D., 72 Pearl Street, Worcester, Mass. Surgeon-in-Chief to Memorial Hospital; Consulting Surgeon to Worcester City and St. Vincent's Hospitals.

ELECTED

1910. Gaub, Otto Carl, M.D., F.A.C.S. Hotel Schenley, Pittsburgh, Pa. Surgeon to Allegheny General Hospital and to Columbia Hospital.

†1882. Gay, George Washington, A.M., M.D., 665 Boylston Street, Boston, Mass. Senior Surgeon to the Boston City Hospital. *Member of Council,* 1882.

†1890. Gerster, Arpad G., M.D., Chir.D., 34 East Seventy-fifth Street, New York. Emeritus Professor of Surgery in the New York Polyclinic, former Professor of Clinical Surgery, Columbia University; Consulting Surgeon to Lenox Hill and Mount Sinai Hospitals.

1906. Gibbon, John H., M.D., *Secretary,* 1608 Spruce Street, Philadelphia. Professor of Surgery, Jefferson Medical College and Surgeon to the Jefferson College Hospital; Surgeon to the Pennsylvania Hospital and Consulting Surgeon to the Bryn Mawr Hospital.

1906. Gibson, C. L., M.D., 72 East Fifty-fourth Street, New York, N. Y. Professor of Surgery, Cornell University Medical College; Attending Surgeon to the First Surgical (Cornell Medical) Division of the New York Hospital; Consulting Surgeon to St. Luke's, City, and General Memorial Hospitals. *Vice-President,* 1915.

1920. Graham, Evarts A., A.B., M.D., Euclid Avenue and Kingshighway, St. Louis, Mo. Professor of Surgery, Washington University Medical School, St. Louis, Missouri; Surgeon-in-Chief to Barnes Hospital and St. Louis Children's Hospital; Consulting Surgeon, City Hospital No. 20.

1911. Greenough, Robert B., A.B. (Harvard), M.D. (Harvard), 8 Marlborough Street, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital.

1916. Haggard, William D., M.D., F.A.C.S., 706 Church Street, Nashville, Tenn. Professor of Surgery, Vanderbilt University; Surgeon to St. Thomas' Hospital.

1909. Halstead, Albert E., M.D., 30 N. Michigan Avenue, Chicago, Ill. Professor of Surgery, School of Medicine, University of Illinois; Attending Surgeon to the St. Luke's and Cook County Hospitals; Consulting Surgeon to the Illinois Charitable Eye and Ear Infirmary; Lieut.-Col., M.C., U.S.A.

†1892. Halsted, William Stewart, A.B. (Yale University, 1874), M.D. (Columbia University, College of Physicians and

ELECTED

Surgeons, N. Y., 1877), LL.D. (Yale University, 1904, and University of Edinburgh, 1905), Sc.D. (Columbia University, 1904), 1201 Eutaw Place, Baltimore, Md. Foreign Corresponding Member, Harveian Society, London; Honorary Fellow, Royal College of Surgeons, England, 1900; Edinburgh, 1905 and College of Surgeons of America, 1913; Membre corresp. étranger de la société de chirurgie, Paris, 1909; Membre titulaire de l'association française de chirurgie, 1914; Ehrenmitglied der deutschen Gesellschaft für Chirurgie, 1914; Hon. Member Societas medicorum Sverana, Stockholm, 1918; Foreign member Royal Swedish Academy of Science, 1918; Member of the National Academy of Sciences; Surgeon-in-Chief, the Johns Hopkins Hospital; Professor of Surgery and Director of the Surgical Clinic of the Johns Hopkins University.

1909. Hamann, Carl A., M.D., 416 Osborn Bldg., Cleveland, Ohio. Professor of Applied Anatomy, Western Reserve University; Visiting Surgeon to Charity and City Hospitals.

1900. Harris, Malcolm L., M.D., 25 E. Washington St., Chicago, Ill. Professor of Surgery, Chicago Policlinic; Attending Surgeon, Alexian Brothers' Hospital, Henrotin Hospital, Passavant Hospital; Consulting Surgeon to Maurice Porter Hospital for Children.

†1895. Harte, Richard H., M.D., C.M.G., 1503 Spruce Street, Philadelphia. Emeritus Surgeon to the Pennsylvania Hospital and Consulting Surgeon to St. Mary's, St. Timothy's, Abington, and Bryn Mawr Hospitals; Colonel M.R.C., U.S.A; formerly Director Base Hospital No. 10, U.S.A., and later O. C. No. 16, General Hospital, B.E.F. *President*, 1910.

1916. Hartwell, John A., Ph.B., M.D., 27 East Sixty-third Street, New York. Associate Professor of Surgery and Professor of Clinical Surgery, Cornell University Medical College; Director of Surgery, Cornell University Division, Bellevue Hospital; Consulting Surgeon to Lincoln Hospital, Lawrence Hospital, Bronxville, N. Y., and United Hospital, Port Chester, N. Y.

1918. Hodge, Edward Blanchard, A.B., M.D., 2019 Spruce St., Philadelphia, Pa. Surgeon to Presbyterian and Children's Hospitals; Out-patient Surgeon to Pennsylvania Hospital; Associate Surgeon to Widener Memorial School.

1909. Hotchkiss, Lucius Wales, A.B., M.D., 11 East Forty-eighth Street, New York. Professor of Clinical Surgery, Columbia

ELECTED

University; Consulting Surgeon to Bellevue, Knickerbocker, Flushing and Greenwich Hospitals; Junior Surgeon to Roosevelt Hospital, New York City.

1918. Hubbard, Joshua C., M.D., 86 Bay State Road, Boston, Mass. Visiting Surgeon, Boston City Hospital; Surgeon, Newton Hospital; Consulting Surgeon, Norwood Hospital and Leonard Morse Hospital.

†1901. Huntington, Thomas W., A.B., M.D., LL.D., 960 Mills Building, San Francisco, California. Professor of Clinical and Operative Surgery (Emeritus), College of Medicine, University of California; Consulting Surgeon, St. Luke's Hospital, San Francisco; Chief Surgeon, Western Pac. R. R. *Vice-President*, 1906; *President*, 1917.

1919. Hupp, Frank Le Moyne, A.M., M.D., 61 Fourteenth St., Wheeling, W. Va. Attending Surgeon, Ohio Valley General Hospital; Consulting Surgeon, Reynolds Memorial Hospital, Glendale; Sheltering Arms Hospital, Hansford.

1913. Hutchison, James Alexander, M.D., L.R.C.P. and S. (Edin.), 354 MacKay Street, Montreal. Professor of Surgery and Clinical Surgery, McGill University; Attending Surgeon, Montreal General Hospital.

1909. Hutchinson, James P., M.D., 133 South Twenty-second Street, Philadelphia. Surgeon to the Pennsylvania, Methodist, Children's, St. Timothy's and Bryn Mawr Hospitals.

1913. Jackson, Jabez N., A.M., M.D., 425 Argyle Building, Kansas City, Mo. Surgeon and Chief of Staff of Christian Church Hospital; Surgeon, Kansas City General Hospital.

†1905. Johnson, Robert W., A.B., M.D., 101 W. Franklin Street, Baltimore, Md. Professor of Principles and Practice of Surgery, Baltimore Medical College; Surgeon to Maryland General Hospital, Church Home and Infirmary, and St. Agnes' Hospital. Retired.

1901. Jonas, A. F., M.D., 512 McCayne Bldg., Omaha, Nebraska. Professor of Surgery, Omaha Medical College; Surgeon to the Methodist Episcopal Hospital and to the Douglas County Hospital; Captain, M.C. *Vice-President*, 1906.

1917. Jones, Daniel Fiske, A.B., M.D., 195 Beacon Street, Boston, Mass. Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon to the New England Hospital for Women and Children, Brockton City Hospital, Union

ELECTED

Hospital of Fall River, Sturdy Hospital of Attleboro and the Addison Gilbert Hospital of Gloucester, Mass.

1910. JOPSON, JOHN HOWARD, M.D., *Recorder*, 1824 Pine Street, Philadelphia, Pa. Professor of Surgery, the Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, University of Pennsylvania; Surgeon to Presbyterian Hospital and Children's Hospital; Consulting Surgeon to Bryn Mawr Hospital and Philadelphia Home for the Incurables.

1914. JUDD, EDWARD STARR, M.D., Rochester, Minn., Surgeon to Mayo Clinic.

†1899. KAMMERER, FREDERIC, M.D., 51 East Sixty-sixth Street, New York City. Professor of Clinical Surgery, Columbia University; Attending Surgeon to the Lenox Hill Hospital; Consulting Surgeon to St. Francis Hospital, New York.

1913. KANAVEL, ALLEN B., A.B., M.D., 30 North Michigan Avenue, Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Attending Surgeon to Wesley Memorial and Cook County Hospitals.

*†1880. KEEN, WILLIAM WILLIAMS, A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. Ph.D. (Upsala), Hon. Sc.D. (Jeff. and Harv.), Hon. F.A.C.S., 1729 Chestnut Street, Philadelphia. Emeritus Professor of Surgery in the Jefferson Medical College; Consulting Surgeon to the Jefferson Medical College Hospital and to the Woman's and St. Agnes' Hospitals; Membre Correspondant Etranger de la Société de Chirurgie de Paris; Membre Honoraire de la Société Belge de Chirurgie; Honorary Member, Clinical Society of London, etc. *President*, 1898; *Vice-President*, 1892; *Member of Council*, 1899–1901.

1916. LAW, ARTHUR AYER, M.D., F.A.C.S., 420 Syndicate Building, Minneapolis, Minn. Associate Professor of Surgery, University of Minnesota Medical School, and Mayo Foundation; Surgeon to the Northwestern Hospital; Surgeon to and Assistant Chief of the University Hospital; Lieut.-Col., M.C. (discharged), Commanding University of Minnesota Base Hospital No. 26 in A.E.F.

1901. LE CONTE, ROBERT G., A.B., M.D., 2000 Spruce Street, Philadelphia. Surgeon to the Pennsylvania Hospital; Consulting Surgeon to the Germantown, the Bryn Mawr and the Gynecean Hospitals. *President*, 1915.

ELECTED

1910. Lee, Burton James, Ph.B., M.D., F.R.C.S., 128 East 73d Street, New York. Clinical Professor of Surgery, Cornell Medical School; Associate Attending Surgeon, New York Hospital; Consulting Surgeon, Sharon (Connecticut) Hospital.

1912. Lewis, Dean D., M.D., 122 S. Michigan Ave., Chicago, Ill. Professor of Surgery, Rush Medical College; Attending Surgeon to the Presbyterian Hospital, Chicago; Consulting Surgeon to the Annie Durand Hospital for Contagious Diseases.

1910. Lilienthal, Howard, A.B., M.D., 52 East Eighty-second Street, New York. Attending Surgeon, Mount Sinai Hospital; Visiting Surgeon to Bellevue Hospital; Consulting Surgeon, Hospital for Joint Diseases, Jewish Maternity Hospital and Har Moriah Hospital; Professor of Clinical Surgery, Cornell Medical College; formerly Lieut.-Col., M.C., U.S.A.

1908. Lothrop, Howard Augustus, A.M., M.D., 101 Beacon St., Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Surgeon-in-Chief, Boston City Hospital.

1916. Lower, William E., M.D., Euclid Avenue and 93d Street, Cleveland, Ohio. Associate Professor of Genito-urinary Surgery, School of Medicine, Western Reserve University; Director of Surgery, Mount Sinai Hospital; Associate Surgeon in charge of Genito-urinary Surgery, Lakeside Hospital; Visiting Surgeon, Lutheran Hospital and White Hospital (Ravenna).

1910. Lund, Frederick Bates, M.D., 527 Beacon Street, Boston, Mass. Associate in Surgery, Harvard Medical School; Surgeon-in-Chief, Boston City Hospital; Consulting Surgeon, Burbank Hospital, Fitchberg, Newton Hospital, Newton; lately Lieut.-Col., M.C., U.S.A.

1917. Lyle, Henry H. M., M.D., 117 East Fifty-sixth Street, New York City. Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Attending Surgeon, St. Luke's Hospital.

1901. McArthur, L. L., M.D., 122 S. Michigan Boulevard, Chicago, Ill. Professor of Clinical Surgery, Post-Graduate Medical School; Assistant Professor of Clinical Surgery, Rush Medical College; Senior Surgeon to St. Luke's and Michael Reese Hospitals.

ELECTED

‡1882. McGRAW, THEODORE A., M.D., 73 Case Street, Detroit, Mich. Professor of Surgery in the Detroit Medical College; Surgeon to St. Mary's and Harper Hospitals. *Vice-President*, 1896.

1917. McGUIRE, STUART, M.D., LL.D., 513 East Grace Street, Richmond, Va. Professor of Surgery, Medical College of Virginia; Surgeon in Charge of St. Luke's Hospital; Visiting Surgeon to Memorial Hospital.

†1903. McMURTRY, LEWIS S., A.M., M.D., LL.D., 542 Atherton Building, Louisville, Ky. Professor of Gynecology and Abdominal Surgery in the Medical Department, University of Louisville; Surgeon to the Louisville City Hospital.

‡1915. McRAE, FLOYD W., M.D., Peters Building, Atlanta, Ga. Chief Surgeon to the Piedmont Sanatorium; Visiting Surgeon to St. Joseph's Infirmary; Consulting Surgeon to Grady Hospital.

1918. McWILLIAMS, CLARENCE A., M.A., M.D., 19 East Sixty-fifth Street, New York, N. Y. Associate Surgeon, Presbyterian Hospital, Instructor in Surgery, Columbia University.

1904. MACLAREN, ARCHIBALD, M.D., B.S., 914 Lowry Building, St. Paul, Minn. Professor of Clinical Surgery, University of Minnesota. *Vice-President*, 1915.

1914. MACLAREN, MURRAY, B.A., M.B.C.M., M.D., M.R.C.S. (Eng.), C.M.G. (Order of Avis), LL.D., 75 Coburg Street, St. John's, N.B., Canada. Surgeon to the General Public Hospital; Colonel, C.A.M.C.

1920. MAES, URBAN, M.D., 1671 Octavia Street, New Orleans, Louisiana. Professor of Clinical and Operative Surgery, Tulane University of Louisiana School of Medicine; Junior Surgeon, Charity Hospital, Touro Infirmary; Senior Surgeon, New Orleans Dispensary for Women and Children.

1898. MARTIN, EDWARD, A.M., M.D., LL.D., 135 S. 18th Street, Philadelphia. Commissioner of Health of Pennsylvania; Professor of Surgical Physiology, University of Pennsylvania; Consulting Surgeon to State Hospitals, Norristown and Wernersville, Pa.; Colonel, M.R.C., U.S.A.

1915. MARTIN, WALTON, Ph.B., M.D., 151 East Sixty-third Street, New York. Professor of Clinical Surgery, Columbia University; Attending Surgeon, St. Luke's Hospital; Consulting Surgeon, White Plains Hospital and Loomis Sanatorium.

†1895. MASTIN, WILLIAM McDOWELL, M.D., LL.D., Northeast corner of Joachim and Conti Streets, Mobile, Ala. Surgeon to the Mobile City Hospital and Providence Infirmary.

ELECTED

1895. Matas, Rudolph, M.D., LL.D., 2255 St. Charles Avenue, New Orleans, Louisiana. Professor of Surgery, Tulane University of Louisiana, School of Medicine; Professor (Emeritus), Graduate Medical School, Tulane University; Senior, Surgical Division, Touro Infirmary; Chief of Surgical Service, Visiting Staff, Charity Hospital; Consulting Surgeon, Eye, Ear, Nose and Throat Hospital; Major, Reserve Corps, U. S. Army. *President*, 1909; *Vice-President*, 1901.

1918. Mathews, Francis Stuart, B.S., M.D., F.A.C.S., 62 West Fiftieth Street, New York. Professor of Clinical Surgery, Columbia University; Surgeon to St. Luke's Hospital and St. Mary's Hospital for Children.

1903. Mayo, Charles H., A.M., M.D., LL.D., D.Sc., F.A.C.S., Rochester, Minn. Professor of Surgery, Medical School, University of Minnesota; Surgeon to Mayo Clinic (St. Mary's Hospital).

1899. Mayo, William J., A.M., M.D. (Mich.), D.Sc. (Columbia and Mich.), F.R.C.S. (Eng. and Edin.), LL.D. (Tor., Md. and Penna.), C.E., Soc. de Chir. de Paris, F.A.C.S., 701 College Hill, Rochester, Minn. Surgeon to St. Mary's Hospital. *Vice-President*, 1903; *President*, 1915.

1901. Meyer, Willy, M.D., 700 Madison Avenue, New York. Professor of Surgery in the New York Post-Graduate Medical School and Hospital; Attending Surgeon to the Lenox Hill Hospital; Consulting Surgeon to the New York Infirmary for Women and Children, New York Skin and Cancer Hospital, Hospital for Deformities and Joint Diseases and the Montefiore Hospital.

1919. Miller, Robert Talbott, A.B., M.D., Duxbury, Mass.

1917. Mitchell, Charles F., M.D., 332 South Fifteenth Street, Philadelphia, Pa. Attending Surgeon to the Germantown, Bryn Mawr and Pennsylvania Hospitals.

1912. Mitchell, James F., A.B., M.D., 1344 Nineteenth Street, Washington, D. C. Clinical Professor of Surgery, Georgetown and George Washington Universities; Chief Surgeon, Emergency Hospital. *Vice-President*, 1920.

†1893. Mixter, Samuel Jason, S.B. (Massachusetts Institute of Technology), M.D. (Harvard), 180 Marlborough Street, Boston. Consulting Surgeon to Massachusetts General Hospital and to Massachusetts Charity Eye and Ear Infirmary. *President*, 1916.

ELECTED

1920. Mixter, William Jason, S.B., M.D., F.A.C.S., 180 Marlborough Street, Boston, Massachusetts. Assistant Visiting Surgeon, Massachusetts General Hospital; Assistant in Surgery, Graduate School of Medicine, Harvard University; Surgeon to the Staff, Anna Jaques Hospital, Newburyport; Consulting Surgeon, Leonard Morse Hospital, Natick, Charitable Eye and Ear Infirmary, Boston, Cable Memorial Hospital, Ipswich; Ex. Lieut.-Col., M.C., U.S.A.

†1896. Monks, George Howard, A.B. (Harvard), M.D. (Harvard), M.R.C.S. (Eng.), 51 Commonwealth Ave., Boston. Consulting Surgeon, Boston City Hospital; Professor of Oral Surgery, Harvard Dental School.

1914. Moschcowitz, Alexis V., M.D., 925 Madison Ave., New York, N. Y. Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Attending Surgeon, Mount Sinai Hospital.

1904. Mudd, Harvey G., M.D., Humboldt Bldg., St. Louis, Mo. Clinical Professor of Surgery, Medical Department, Washington University; Surgeon to St. Luke's Hospital; Consulting Surgeon to the Barnard Free Skin and Cancer Hospital. *Vice-President*, 1920.

1919. Müller, George P., M.D., 1930 Spruce St., Philadelphia, Pa. Professor of Surgery, Graduate School of the University of Pennsylvania; Associate in Surgery, Medical School of the University of Pennsylvania; Surgeon to the Misericordia and St. Agnes Hospitals; Consulting Surgeon to the Chester County Hospital, West Chester, Pa.

1913. Murphy, Fred T., A.B., M.A., M.D., 2248 Penobscot Bldg., Detroit, Michigan.

†1898. Murray, Francis W., M.D., 32 West Thirty-ninth Street, New York City. Consulting Surgeon, New York Hospital, St. Luke's Hospital, New York Eye and Ear Infirmary, Paul Kimball Hospital, Lakewood, Goshen Emergency Hospital.

1903. Neilson, Thomas R., M.D., 1937 Chestnut Street, Philadelphia, Pa. Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Clinical Professor of Genito-urinary Diseases in the University of Pennsylvania.

ELECTED

1915. Nichols, Edward Hall, A.B., A.M., M.D., 294 Marlborough Street, Boston, Massachusetts. Clinical Professor of Surgery, Harvard University Medical School; Senior Visiting Surgeon, Boston City Hospital; Consulting Surgeon, City Hospital, Quincy, and Framingham Hospital, Framingham.

1900. Ochsner, A.J., B.S., F.R.M.S., M.D. (Eng.), LL.D.; F.A.C.S., 710 Sedgwick Street, Chicago. Professor of Clinical Surgery, College of Physicians and Surgeons; Surgeon-in Chief of Augustana and St. Mary's Hospitals.

1900. Oliver, J. C., M.D., 628 Elm Street, Cincinnati, Ohio. Acting Dean and Professor of Surgery, Medical Department, University of Cincinnati; Director of Second Surgical Service, Cincinnati General Hospital; Surgeon to the Christ Hospital.

1915. Olmsted, Ingersoll, M.B., 215 James Street, South, Hamilton, Ontario. Surgeon to City Hospital.

†1882. Owens, John E., M.D., 1017–18 Venetian Building, Chicago, Ill. Professor of Principles and Practice of Surgery and Clinical Surgery in the Chicago Medical College; Surgeon to St. Luke's Hospital. *Vice-President*, 1900.

1899. Parham, Frederick William, M.D., 3513 Prytania Street, New Orleans, La. Professor of General Clinical Surgery, New Orleans Polyclinic (Post-Graduate Department of Tulane University of Louisiana); Chief of Staff to Charity Hospital and to Touro Infirmary.

†1893. Parmenter, John, M.D., 519 Franklin Street, Buffalo, N. Y. Late Professor of Clinical Surgery, Medical Department, University of Buffalo.

1908. Peck, Charles Howard, *Treasurer*, M.D., 30 W. Fiftieth St., New York City. Professor of Clinical Surgery, Columbia University; Senior Attending Surgeon to the Roosevelt Hospital; Consulting Surgeon to French Hospital, Memorial Hospital and Hospital for Ruptured and Crippled, New York City; Vassar Brothers Hospital, Poughkeepsie, N. Y.; Nyack Hospital, Nyack, N. Y.; White Plains Hospital, White Plains, N. Y.; United Hospital, Portchester and Rye, N. Y.; Stamford Hospital, Stamford, Conn.; Greenwich Hospital, Greenwich, Conn.; Hackensack Hospital, Hackensack, N. J.

ELECTED

1919. Percy, Nelson Mortimer, M.D., F.A.C.S., 2106 Sedgwick Street, Chicago, Ill. Associate Professor of Clinical Surgery, Medical Department of the University of Illinois; Attending Surgeon to the Augustana and St. Mary's of Nazareth Hospitals.

1917. Phemister, Dallas B., B.S., M.D., 122 South Michigan Ave., Chicago, Ill. Assistant Professor in Surgery, Rush Medical College; Associate Attending Surgeon, Presbyterian Hospital.

†1889. Pilcher, Lewis Stephen, A.M., M.D., LL.D. (Dickinson and University of Michigan), 145 Gates Avenue, Brooklyn, N. Y. Consulting Surgeon to the St. John, Norwegian, Jewish, Bushwick and Deaconess Hospitals. *Vice-President*, 1893, 1914. *President*, 1918.

1914. Plummer, Samuel C., A.B., M.D., 4539 Oakenwald Avenue, Chicago, Ill. Attending Surgeon, St. Luke's Hospital; Chief Surgeon, C. R. I. & P. Ry. Co.

1914. Pool, Eugene Hillhouse, A.B., M.D., 107 East Sixtieth Street, New York, N. Y. Attending Surgeon, New York Hospital; Associate Professor of Surgery, Columbia University; Consultant Surgeon to French Hospital, New York Orthopedic, Manhattan State Hospital, Home for Incurables and United Hospital at Portchester.

1904. Porter, Charles Allen, A.M., M.D., 116 Beacon Street, Boston, Mass. Chief of West Surgical Service, Massachusetts General Hospital; Professor of Clinical Surgery, Harvard Medical School.

1915. Porter, Miles Fuller, M.A., M.D., Walnut Place, Fort Wayne, Ind. Professor of Surgery in the Indiana University School of Medicine; Surgeon to St. Joseph's Hospital; Consulting Surgeon in the Indiana School for Feeble-minded Youths.

†1896. Powers, Charles A., A.M., M.D., University Club, Denver, Col. Professor of Surgery, Emeritus, in the University of Colorado; Consulting Surgeon to the Denver City and County Hospital, to the Mercy Hospital, and to the State Home for Children. *President*, 1912; *Vice-President*, 1903; *Treasurer*, 1906–1912; *Member of Council*, 1906–1916.

1908. Primrose, Alexander, C.B., M.B., C.M. (Edin.), M.R.C.S. (Eng.). *Vice-President.* 100 College Street, Toronto, Canada. Surgeon to the Toronto General Hospital and Consulting Surgeon to the Hospital for Sick Children, Toronto; Professor of Clinical Surgery in the University of Toronto.

ELECTED

1901. Rixford, Emmet, B.S., M.D., 1705 California Street, San Francisco, Cal. Professor of Surgery in Stanford University School of Medicine; Surgeon to the San Francisco and Lane Hospitals; Member of the Society of Clinical Surgery. *Vice-President*, 1905.

†1882. Roberts, John B., A.M., M.D., 313 South Seventeenth Street, Philadelphia. Professor of Surgery in the University of Pennsylvania Graduate School of Medicine. *Vice-President*, 1888 and 1894; *Treasurer*, 1892–93; *President*, 1920.

1914. Ross, George Gorgas, M.D., 1721 Spruce Street, Philadelphia, Pa. Visiting Surgeon, Germantown Hospital; Assistant Surgeon, Lankenau Hospital; Visiting Surgeon to the Stetson and Methodist Hospitals; Assistant Surgeon to the University Hospital; Associate in Surgery, University of Pennsylvania.

1917. Rowan, Charles J., A.B., M.D., Iowa City, Iowa. Professor and Head of Department of Surgery, College of Medicine, State University of Iowa; Director of the University Hospital.

†1882. Rushmore, John Dikeman, M.D., 129 Montague Street, Brooklyn, N. Y. Professor of Operative and Clinical Surgery, Emeritus, Long Island College Hospital; Surgeon to St. Peter's Hospital; Consulting Surgeon to Long Island College Hospital, Kings County Hospital, Swedish and Lutheran Hospitals.

1909. Scudder, Charles L., M.D., A.B., Ph.B., 209 Beacon Street, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Chief of the East Surgical Service, Massachusetts General Hospital; Member of the Administrative Board of the Graduate School of Medicine, Harvard University.

1919. Seelig, M. G., A.B., M.D., 4952 Park View Place, St. Louis, Mo. Professor of Surgery, St. Louis University; Visiting Surgeon to the Jewish Hospital and to the St. Louis City Hospital.

†1902. Shepherd, Francis J., M.D., LL.D., F.R.C.S. (Eng.), 152 Mansfield Street, Montreal, Canada. Emeritus Professor in McGill University; Consulting Surgeon to the Montreal General Hospital and Royal Victoria Hospital, Montreal.

1920. Sistrunk, Walter E., M.D., F.A.C.S., Surgeon to Saint Mary's and the Colonial Hospitals (Mayo Clinic), Rochester, Minn.; Associate Professor of Surgery, Mayo Foundation, University of Minnesota.

ELECTED

1918. Smith, Rea, M.D., 22 Chester Place, Los Angeles, Cal. Surgeon, California Hospital and Los Angeles County Hospital.

†1895. Souchon, Edmond, M.D., 2403 St. Charles Avenue, New Orleans, La. Professor of Anatomy and of Clinical Surgery, Tulane University; Surgeon to Charity Hospital; Consulting Surgeon to the Eye, Ear, Nose, and Throat Hospital. Honorary Fellow of American College of Surgeons; Former President of Louisiana State Board of Health, 1898–1905. *Vice-President*, 1899.

1919. Speed, Kellogg, S.B., M.D., F.A.C.S., 122 South Michigan Avenue, Chicago, Ill. Assistant Professor of Surgery, Rush Medical College, Attending Surgeon, Cook County and Provident Hospitals; Assistant Surgeon, Presbyterian Hospital; Consulting Surgeon, U. S. P. H. S. Hospital No. 2.

1918. Starr, Clarence Leslie, M.B., M.D., 224 Bloor St., West, Toronto, Canada. Associate Professor of Surgery, University of Toronto; Surgeon-in-Chief, Hospital for Sick Children; Consultant in Surgery, Toronto General Hospital; Surgeon to St. John's Hospital; Lieut.-Col.-Consultant, C. A. M. C.

1914. Starr, Frederic Newton Gisborne, C.B.E., M.B., M.D., C.M., 112 College Street, Toronto, Canada. Associate Professor of Clinical Surgery, University of Toronto; Surgeon to Hospital for Sick Children, and to St. John's Hospital for Women; Senior Assistant Surgeon, Toronto General Hospital.

1915. Stewart, George David, M.D., 61 West Fiftieth Street, New York. President, New York Academy of Medicine; Professor of Surgery, New York University Medical School; Visiting Surgeon to St. Vincent's and Bellevue Hospitals; Consulting Surgeon to Hospital for Ruptured and Crippled of New York, to St. Joseph's Hospital, Yonkers, to St. Mary's Hospital, East Orange, N. J., to South Side Hospital, Babylon, L. I., to Englewood Hospital, Englewood, N. J., and to the United Hospital, Portchester, N. Y.

1909. Stillman, Stanley, M.D., 2101 Webster Street, San Francisco, Cal. Professor of Surgery, Stanford University Medical School; Commander, M. C., U. S. N. R. F.; Surgeon to Lane and Stanford University Hospitals.

1912. Stokes, Charles F., A.M., M.D., D.Sc., LL.D., 6 West 77th Street, New York City. Former Surgeon-General, U. S. N.

ELECTED

1900. Summers, John Edward, M.D., 618 Brandeis Theatre Bldg., Omaha, Neb. Professor of Clinical Surgery, University of Nebraska Medical School; Surgeon to the University Hospital and to the Clarkson Memorial, the Wise Memorial, and the Douglas County Hospitals, Omaha, Neb.

1920. Taylor, Alfred S., B.P., A.M., M.D., 115 W. 55th Street, New York. Professor of Clinical Surgery, Cornell Medical School; Visiting Surgeon, Fordham Hospital and Neurological Institute; Neurological Surgeon, Hospital for Ruptured and Crippled; Consulting Surgeon to Memorial Hospital, New York City, Tarrytown Hospital, New York, White Plains Hospital, New York, Overlook Hospital, Summit, N. J.

1900. Taylor, William J., M.D., 1825 Pine Street, Philadelphia. Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes' Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women and to the Woman's Hospital.

1916. Terry, Wallace I., B.S., M.D., 240 Stockton Street, San Francisco, Cal. Professor of Surgery, University of California; Chief Surgeon, University of California Hospital, San Francisco.

1909. Thompson, James E., M.B., B.S. (London), F.R.C.S. (Eng.), F.A.C.S. *Vice-President.* 3224 Broadway, Galveston, Texas. Professor of Surgery in the University of Texas; Surgeon to the John Sealy Hospital.

1920. Tinker, Martin B., B.S., M.D., 101 South Aurora Street, Ithaca, N. Y. Surgeon to the Ithaca City Hospital and the Cornell Infirmary.

1918. Truesdale, Philemon Edwards, M.D., 177 Winter St., Fall River, Mass. Surgeon to St. Anne's and Highland Hospitals; Consulting Surgeon to Fall River City Hospital.

†1882. Vander Veer, Albert, A.M., M.D., Ph.D., LL.D., 28 Eagle Street, Albany, N. Y. Surgeon-in-Chief, Albany Hospital; Consulting Surgeon, Albany Free Dispensary Association, Benedictine Hospital, Kingston, N. Y., and Champlain Valley Hospital, Plattsburgh, N. Y. *President,* 1905; *Vice-President,* 1898; *Member of Council,* 1907.

1910. Vander Veer, Edgar Albert, Ph.B., M.D., 28 Eagle Street, Albany, N. Y. Attending Surgeon, Albany Hospital; Consulting Surgeon, Champlain Valley Hospital and Benedictine Hospital, Kingston, N. Y.

ELECTED

1921. VAUGHAN, J. WALTER, A.B., M.D., 987 East Jefferson Avenue, Detroit, Mich. Associate Attending Surgeon to Harper Hospital.

1902. VAUGHAN, GEORGE TULLY, M.D., LL.D., 1718 I Street, Washington, D. C. Professor of Principles and Practice of Surgery and Head of the Surgical Department, Georgetown University; Chief Surgeon, Georgetown University Hospital; Surgeon, Tuberculosis Hospital; Consulting Surgeon, Government Hospital for the Insane, Washington, D. C., U. S. Public Health Service and Washington Asylum Hospital; Commander, M. C., Reserve Force, U. S. N.

1920. VERDI, WILLIAM FRANCIS, A.M., M.D., 27 Elm Street, New Haven, Connecticut. Clinical Professor of Surgery, Yale University School of Medicine; Chief Surgeon, Hospital of St. Raphael; Attending Surgeon, New Haven Hospital; Consulting Surgeon, St. Vincent's Hospital, Bridgeport, St. Mary's Hospital, Waterbury, Middlesex Hospital, Middletown.

1918. WAINWRIGHT, JONATHAN M., A.B., A.M., M.D., 912 Clay Ave., Scranton, Pa. Surgeon-in-Chief, Moses Taylor Hospitals (Scranton and Buffalo); Chief Surgeon, D. L. & W. R. R. Co.

†1891. WALKER, EDWARD W., M.D., 214 West Seventh Street, Cincinnati, Ohio. Professor of Surgery, Miami Medical College.

1909. WALKER, JOHN B., M.D., 51 East Fiftieth Street, New York City. Surgeon to Bellevue Hospital and Hospital for Ruptured and Crippled, New York; Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University, N. Y.

†1882. WARREN, JOHN COLLINS, M.D., LL.D. (Jefferson, Harvard and McGill), Hon. F.R.C.S. (Eng. and Edin.), 58 Beacon Street, Boston, Mass. Emeritus Professor of Surgery in Harvard University; Consulting Surgeon to the Massachusetts General Hospital. *President*, 1896; *Vice-President*, 1890; *Member of Council*, 1897.

†1896. WATSON, FRANCIS SEDGWICK, A.B., M.D., 92 Marlborough St., Boston. Surgeon to the Boston City Hospital; Instructor in Genito-Urinary Surgery, Harvard Medical School.

1913. WATTS, STEPHEN H., M.A., M.D., Department of Surgery, University of Virginia, Charlottesville, Va. Professor of Surgery and Gynecology in the University of Virginia; Surgeon-in-Chief, University of Virginia Hospital.

†1889. Weir, Robert F., A.M., M.D., Hon. F.R.C.S. (Eng.), Cor. Mem. Surg. Soc., Paris, 1155 Park Avenue, New York. Professor of Surgery in the College of Physicians and Surgeons, Columbia University; Surgeon to the Roosevelt Hospital. *President*, 1899; *Member of Council*, 1893–1896; 1901–03.

†1892. Wharton, Henry R., A.M., M.D., 1725 Spruce Street, Philadelphia. Surgeon to the Presbyterian and Children's Hospitals.

1921. Whipple, Allen O., B.S., M.D., Presbyterian Hospital, 41 East Seventieth Street, New York, N. Y. Professor of Surgery, Columbia University; Director of Surgical Service, Presbyterian Hospital.

1921. Williams, Hugh, A.B., M.D., 301 Beacon Street, Boston, Mass. Assistant, Harvard Graduate School of Medicine; Visiting Surgeon, Massachusetts General Hospital.

1914. Winslow, Randolph, A.B., M.D., A.M., LL.D., 1900 Mt. Royal Terrace, Baltimore, Md. Professor of Surgery, University of Maryland; Chief Surgeon, University Hospital; Consulting Surgeon, Hebrew Hospital, Baltimore, Union Hospital, Elkton, Md., and to James L. Kernan Hospital for Crippled Children, Baltimore.

1918. Wood, Alfred Conard, M.D., 2035 Walnut St., Philadelphia, Pa. Assistant Professor of Surgery, University of Pennsylvania; Surgeon to the University, Philadelphia General, Howard, St. Timothy's and Rush Hospitals.

1901. Woolsey, George, M.D., 117 East Thirty-sixth Street, New York City. Professor of Clinical Surgery, Cornell University; Surgeon to Bellevue Hospital; Consulting Surgeon to Memorial, Peekskill and St. John's Riverside, Yonkers.

1916. Yates, J. L., Ph.B., B.S., M.D., F.A.C.S., 141 Wisconsin Street, Milwaukee, Wis. Chief of Staff, Milwaukee County Hospital; Attending Surgeon, Columbia Hospital.

SENIOR FELLOWS OF THE ASSOCIATION

Abbe, Robert, M.D., 13 West Fiftieth Street, New York.

Barton, James M., A.M., M.D., 32 North New Hampshire Ave., Atlantic City, N. J.

Bradford, E. H., A.B., M.D., 133 Newbury Street, Boston, Mass.

Carmalt, William H., A.M., M.D., 261 St. Ronan St., New Haven, Conn.

Carson, Norman Bruce, M.D., 303 Humboldt Bldg., St. Louis, Mo.

Cushing, Hayward Warren, A.B., M.D., 70 Commonwealth Ave., Boston, Mass.

Dennis, Frederic Shepard, M.D., F.R.C.S. (Eng.), 62 East 55th Street, New York, N. Y.

Elliott, John Wheelock, A.M., M.D., 124 Beacon Street, Boston, Mass.

Estes, William Lawrence, A.M., M.D., South Bethlehem, Pa.

Gay, George W., A.M., M.D., 665 Boylston Street, Boston, Mass.

Gerster, Arpad G., M.D., Chir.D., 34 East Seventy-fifth Street, New York, N. Y.

Halsted, Wm. Stewart, A.B., LL.D., S.C.D., M.D., Hon. F.R.C.S. (Eng. and Edin.), Hon. F.A.C.S., 1201 Eutaw Place, Baltimore, Md.

Harte, Richard H., M.D., C.M.G., 1503 Spruce Street, Philadelphia.

Huntington, Thomas W., A.B., M.D., LL.D., 960 Mills Building, San Francisco, Cal.

Johnson, Robert W., A.B., M.D., 101 W. Franklin St., Baltimore, M.D.

Kammerer, Frederic, M.D., 51 East Sixty-sixth Street, New York City.

Keen, W. W., A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. F.A.C.S., 1720 Chestnut Street, Philadelphia, Pa.

Mastin, Wm. M., M.D., LL.D., Northeast corner Joachim and Conti Streets, Mobile, Ala.

McGraw, Theodore A., M.D., 73 Case Street, Detroit, Mich.

McMurtry, Lewis S., A.M., M.D., LL.D., 542 Atherton Building, Louisville, Ky.

Minter, Samuel Jason, S.B., M.D., 180 Marlborough St., Boston, Mass.

Monks, George H., A.B., M.D., M.R.C.S., 51 Commonwealth Avenue, Boston, Mass.

Murray, Francis W., M.D., 32 West Thirty-ninth Street, New York, N. Y.

Owens, John E., M.D., 1201 Venetian Building, Chicago, Ill.

Parmenter, John, M.D., 519 Franklin Street, Buffalo, N. Y.

Pilcher, Lewis S., A.M., M.D., LL.D., 145 Gates Avenue, Brooklyn, New York. President, 1918.

Powers, Charles A., A.M., M.D., University Club, Denver, Colo.

Roberts, John B., A.M., M.D., 313 South Seventeenth Street, Philadelphia, Pa.

Rushmore, John D., M.D., 129 Montague Street, Brooklyn, N. Y.

Shepherd, Francis J., M.D., LL.D., F.R.C.S. (Eng.), 152 Mansfield Street, Montreal, Canada.

Souchon, Edmund, M.D., 2403 St. Charles Ave., New Orleans, La.

Vander Veer, A., A.M., M.D., Ph.D., LL.D., 28 Eagle Street, Albany, N. Y.

Walker, Edward W., M.D., 214 West Seventh Street, Cincinnati, Ohio.

Warren, J. Collins, M.D., LL.D., Hon. F.R.C.S. (Eng. and Edin.), 58 Beacon Street, Boston, Mass.

Watson, Francis S., A.B., M.D., 92 Marlborough Street, Boston, Mass.

Weir, Robert F., A.M., M.D., Hon. F.R.C.S. (Eng.), 1155 Park Avenue, New York, N. Y.

Wharton, Henry R., A.M., M.D., 1725 Spruce Street, Philadelphia, Pa.

HONORARY FELLOWS OF THE ASSOCIATION

ELECTED

1907. Ballance, Charles Alfred, K.C.M.G., C.B., M.V.O., M.B., M.S., University of London, F.R.C.S. (Eng.), 106 Harley Street, London. Consulting Surgeon to St. Thomas's Hospital and to the National Hospital for Paralysis and Epilepsy, Queen Sq.

1919. Bastianelli, Raffaelle, Visle della Regina 231, Rome, Italy.

1919. Bowlby, Sir Anthony A., K.C.B., K.C.M.G., V.C.V.O., 25 Manchester Square. W., London, England. Consulting Surgeon to St. Bartholomew's Hospital and to Queen Alexandra's Military Hospital.

1891. Chiene, John, F.R.C.S. (Edin.), M.D. (Edin.), Aithernie Davidsons Mains, Midlothian. Emeritus Professor of Surgery, University of Edinburgh.

1916. Depage, Prof. Dr. Antoine, 75 Avenue Louise, Belgium.

1919. Hartmann, Dr. Henri, 4 Place Malesherbes, Paris, France. Professor of Clinical Surgery, Faculté de Médecine de Paris and Surgeon to the Hotel Dieu.

1920. Keen, W. W., A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. Ph.D. (Upsala), Hon. Sc.D. (Jeff. and Harv.), Hon. F.A.C.S., 1729 Chestnut Street, Philadelphia.

1918. Keogh, Sir Alfred, Ky., K.C.B., Royal Army Medical Corps, England.

1894. Macewen, Sir William, M.D., M.B., M.S., 3 Woodside Crescent, Glasgow, Scotland. Professor of Surgery, The University, Glasgow.

1918. Makins, Sir George, Surgeon General, K.C.M.G., C.B., F.R.C.S., England.

1920. Manoury, Gabriel, Dr., Rue Bonneval 26, Chartres, France, Ex-President of French Surgical Association.

1908. Moynihan, Sir B. G. A., M.S. (Lond.), F.R.C.S., 33 Park Square, Leeds, England. Surgeon to the Leeds Infirmary.

ELECTED

1907. Mylius, Sir Thomas, M.D., F.R.C.S.I., 33 Merrion Square, N., Dublin, Ireland.

1903. Robson, A. W. Mayo, C.V.O., M.D., F.R.C.S., D.Sc., 8 Park Crescent, Portland Place, London, W., England. Vice-President, Royal College of Surgeons of England; Hunterian Professor of Surgery and Pathology.

1882. Smith, Stephen, A.M., M.D., LL.D., 574 Madison Avenue, New York. Professor of Clinical Surgery in the University Medical College of New York; Surgeon to the Bellevue and St. Vincent's Hospitals. *Vice-President*, 1890.

1912. Stiles, Sir Harold Jalland, M.B., F.R.C.S. (Edin.), 9 Great Stuart Street, Edinburgh, Scotland. Surgeon to Chalmers Hospital and to the Royal Edinburgh Hospital for Sick Children, Edinburgh.

1914. Thomson, Alexis, B.M., M.S., M.D., (Edin.), M.R.C.S. (Eng.), 39 Drumsheugh Gardens, Edinburgh, Scotland. Professor of Surgery, University of Edinburgh.

1918. Tuffier, Th., M.D., 42 Avenue Gabriel, Paris, France.

1920. Wallace, Sir Cuthbert, K.C.M.G., C.B., 26 Upper Wimpole St., W., London, Eng.; Surgeon to St. Thomas's Hospital, London.

1920. Walther, Charles, M.D., 68 Rue de Belle-Chasse, Paris, France. Professeur Agrégé à la Faculté de Médecine; Chirurgien Honoraire des Hôpitaux; Membre de l'Académie de Médecine.

DECEASED FELLOWS OF THE ASSOCIATION

Name	Date of death
Agnew, D. Hayes	March 22, 1892
Allen, Dudley Peter	January 6, 1915
Allis, Oscar Huntington	May 16, 1921
Annandale, Thomas	1907
Ashhurst, John, Jr.	July 7, 1900
*Atlee, John Light	October 1, 1885
Beckman, Emil Hessel	November 7, 1916
Benham, Silas Nelson	November 3, 1890
*von Bergmann, Ernst	Died since 1906 Meeting
*Bigelow, Henry Jacob	October 30, 1890
*Billings, John Shaw	March 11, 1913
*Billroth, Albert Christian Theodor	January 5, 1894
Bontecou, R. B.	1906–1907
Bosher, Lewis C.	September 12, 1920
Briggs, William Thompson	June 13, 1894
Brinton, John Hill	March 18, 1908
Bristow, Algernon Thomas	March 26, 1913
Brock, Hugh W.	April 24, 1882
Bryant, Joseph Decatur	April 7, 1914
*Bryant, Thomas	1914–1915
Bull, William Tillinghast	February 22, 1909
Burrell, Herbert Leslie	1910
Byrd, William Andrew	August 14, 1887
Cabot, Arthur Tracy	November 4, 1912
Campbell, Henry Fraser	December 15, 1891
Cheever, David Williams	December 27, 1915
Cole, Charles Knox	February 27, 1920
Conner, Phineas Sanborn	March 26, 1909
Cunningham, Francis Deane	September 11, 1885
*Czerny, Prof. Dr. Vincenz	October 3, 1916
Dandridge, Nathaniel P.	1911–1912
Davis, Gwilym G.	June 16, 1918

* Honorary.

Name	Date of death
Davis, John Staige	July 18, 1885
Dawson, William Wirt	February 16, 1893
Dugas, Louis Alexander	October 19, 1884
Dunn, James Henry	June 16, 1904
Dunott, Thomas Justus	May 20, 1893
*Durham, Arthur Edward	May 7, 1895
*Erichsen, Sir John Eric	September 23, 1896
*von Esmarch, Friedrich	1907
Fenger, Christian	March 7, 1902
Ferguson, Alexander Hugh	October 20, 1911
Forbes, William Smith	December 17, 1905
Fowler, George Ryerson	February 6, 1906
*Friedrich, Paul Leopold	January 5, 1916
Gaston, James McFadden	November 17, 1903
Gerrish, Frederic Henry	September 8, 1920
*Gorgas, William C.	July 4, 1920
Greene, W. Warren	September 10, 1881
Gross, Samuel David	May 6, 1884
Gross, Samuel Weissel	April 16, 1889
Gunn, Moses	November 4, 1887
*Gussenbauer, Karl	June 19, 1903
Guthrie, George Washington	June 1, 1915
Harrington, Francis B.	1914–1915
*Harrison, Reginald	1907
Hearn, W. Joseph	December 20, 1917
Hodge, H. Lenox	June 16, 1881
Hodgen, John T.	April 28, 1882
Homans, John	February 7, 1903
*Horsley, Sir Victor Alexander, C. B.	July 16, 1916
Horwitz, Orville	January 28, 1913
*Horwitz, Phineas J.	September 28, 1904
Hughes, J. C.	August 10, 1881
*Humphrey, Sir George Murray	September 24, 1896
*Hunt, William	January 5, 1897

* Honorary.

Name	Date of death
Jacobson, Nathan	September 16, 1913
Johnson, Alexander B.	September 4, 1917
Johnston, George Ben	December 20, 1916
Johnston, Christopher	October 11, 1891
King, Alfred	June 4, 1916
Kinloch, Robert Alexander	December 23, 1891
*Kocher, Theodor	July 27, 1917
Lane, Levi Cooper	February 18, 1902
*von Langenbeck, Bernhardt	September 30, 1887
Levis, Richard J.	November 12, 1890
*Lister (Lord), Joseph	February 11, 1912
Little, James Lawrence	April 4, 1885
Lutz, Frank Joseph	March 24, 1916
McCann, James	1893
McCosh, Andrew J.	December 3, 1908
McGuire, Hunter	September 19, 1900
*MacCormac, Sir William	December 4, 1901
MacDonald, Willis Goss	December 30, 1910
Mackenzie, Kenneth Alexander James	March 15, 1920
MacMonagle, Beverly	May 22, 1912
Markoe, Francis Hartman	1907
Marks, Solon	1916
Mastin, Claudius Henry	October 3, 1898
Mears, J. Ewing	May 28, 1919
Michael, J. Edwin	December 7, 1895
*Mikulicz-Radecki, John V.	
Miles, Albert B.	August 5, 1894
Millard, Perry H.	February 1, 1897
Miller, Truman W.	May 31, 1900
*Moore, Edward Mott	March 3, 1902
Moore, James E.	November 2, 1918
Mudd, Henry Hodgen	November 20, 1899
Mumford, James Gregory	October 18, 1914
Munro, John C.	December 6, 1910
Murphy, John B.	August 11, 1916
de Nancrède, C. B. G.	April 12, 1921
Norris, Basil E.	October, 1895
*von Nussbaum, J. N.	October 31, 1890

* Honorary.

Name	Date of death
*Ollier, Leopold	1900–1901
Oviatt, Charles W.	October 31, 1912
*Paget, Sir James	December 30, 1899
Pancoast, William H.	January 5, 1897
Park, Roswell	February 15, 1914
*Parker, Willard	April 25, 1884
Parkes, Charles T.	March 28, 1891
Parkhill, Clayton	January 16, 1902
*Péan, Jules E.	January 30, 1898
Peck, Washington F.	December 30, 1891
Pilcher, Paul Monroe	January 4, 1917
Porter, Charles Burnham	May 21, 1909
Porter, William Gibbs	January 30, 1906
*Pozzi, Samuel	1918
Prewitt, Theodore F.	October 17, 1904
Prince, David	December 19, 1889
Ransohoff, Joseph	March 10, 1921
Reed, Thomas Baird	April 1, 1891
Richardson, Maurice Howe	July 31, 1912
Richardson, Tobias Gibson	May 26, 1892
Rodman, William Louis	March 8, 1915
Sabine, Thomas Taunton	August 23, 1888
Sayre, Lewis A.	1900–1901
*Schede, Max	1902
Senn, Nicholas	January 2, 1908
Sherman, Harry M.	1921.
*Sims, J. Marion	November 13, 1883
Squire, Truman Hoffman	November 27, 1889
Stewart, Francis T.	February 4, 1920
*Terrier, Louis Felix	1908
*Thiersch, Karl	April 28, 1895
Thompson, Joseph Ford	February 1, 1917
Thompson, Joseph W.	March 30, 1886
Thomson, William	August 3, 1907
Tiffany, Louis McLane	October 23, 1916

* Honorary.

Name	Date of death
Varick, Theodore Romeyn	November 23, 1887
*Verneuil, Aristide A. S.	June 11, 1895
*von Volkmann, Richard	November 28, 1889
Watson, Beriah Andrew	December 22, 1892
Weeks, Stephen Holmes	September 1, 1909
Weist, Jacob Rowland	May 14, 1900
*Wells, Sir Thomas Spencer	January 31, 1897
White, James William	April 24, 1916
Wight, Jarvis S.	November 16, 1901
Willard, DeForest	October 14, 1910
Wood, James R.	May 4, 1882
*Yandell, David Wendell	May 2, 1898

* Honorary.

REPORT OF COMMITTEE ON NECROLOGY

DR. JOSEPH RANSOHOFF

Dr. Joseph Ransohoff, who was born in Cincinnati, May 26, 1853, and died March 10, 1921, gave to his native city all of his life efforts. His early education was obtained in the schools of Cincinnati. He graduated from Woodward High School in the class of 1870, and like many of his contemporaries did not have a university education. He received his medical degree from the Medical College of Ohio in 1874.

He spent the year following his graduation in the Cincinnati Hospital as an intern. The three following years were spent in Europe developing and expanding his medical knowledge, at which time he became M.R.C.S. of England; later the Fellowship was conferred on him. In 1877 he was appointed demonstrator of anatomy in the Medical College of Ohio and served in that position until 1900.

He soon became prominent as an excellent teacher of anatomy. At the same time he began his surgical career. His knowledge of anatomy aided him materially in his surgical work, and he soon became well known as a brilliant surgeon. In 1900 he was made professor of surgery in the Medical College of Ohio. When the Medical College of Ohio united to become the Medical College of the University of Cincinnati in 1909 he was made professor of surgery in the latter institution and served as such until the time of his death.

Although Dr. Ransohoff was eminent as an anatomist and as a surgeon, his claim to preëminence rests upon his unusual ability as a clinical teacher. Here he was at his best, and he had but few rivals. His power of expression and his power to lead students to think marked him as an extraordinary teacher. He had the faculty of so asking questions as to lead the student, almost unwittingly, to a diagnosis from which he could not escape. This ability to implant ideas in the minds of the students was a very exceptional one—an ability possessed by but few medical teachers—it constituted his greatest claim to fame.

Next to his ability as a teacher came his facility of putting his ideas into written words. His writings have an elegance of ex-

pression, an unusual ability in the choice of words and a clearness of thought that mark him as a great writer. Along with his ability to express himself clearly went many of the flowers of rhetoric. He embellished his writings and presented them as a polished, finished product. A touch of the beautiful illumined the dry details of facts.

Dr. Ransohoff led a very busy life, but his efforts were devoted almost entirely to his practice and to the teaching of anatomy and surgery.

Dr. Ransohoff had a long and prosperous career and served his college with marked devotion. He always considered it a sacred duty to fulfil his obligations to the students, and they responded with enthusiasm to his devotion to duty.

His family consisted of his wife, three daughters and two sons, who survive him. His home life was ideal.

The American Surgical Association, to which he was elected to membership in 1886, has lost an able, loyal and faithful member.

J. C. O.

DR. LEWIS CRENSHAW BOSHER

Dr. Lewis Crenshaw Bosher, an outstanding figure in the Southern surgical world, and a Fellow of this Association whose force and charm of personality won him the consideration of its entire membership, died on September 12, 1920, at his home in Richmond, Va., aged sixty-one years.

Though he had been in failing health for some years, and at one time went through a serious spell of sickness, Dr. Bosher had regained his health sufficiently to permit his return to practice in a limited degree. On the morning of his death he was on the streets of the city and attended a patient. During the preceding week he had complained of some indisposition, but had apparently fully recovered from this. Shortly after 7 o'clock in the evening he arose from the dinner table and retired to his bedroom. A few minutes later he was striken with cardiac collapse, secondary to the rheumatism which had troubled him for years. A hurried consultation was held between a dozen different local physicians and surgeons, but nothing could be done to stay the hand of death, which swiftly fell.

Born in Richmond, Va., February 17, 1860, the son of Robert H. and Elizabeth Eubank Bosher, Dr. Bosher, after the usual preliminary education, attended Richmond College, where he received his A.B., degree. In 1883 he was graduated from the Medical College of Virginia. After a year's internship in the City Hospital

he went to New York and spent some time at Mt. Sinai and Bellevue Hospitals, later returning to Richmond to engage in the active practice of surgery. He immediately became identified with the teaching staff of his alma mater and subsequently occupied a position of great influence in its faculty, filling successively the chairs of anatomy, principles of surgery, surgery and genito-urinary surgery.

His striking success as a teacher was equaled by the signal service he rendered in developing and extending the hospital facilities of Richmond. The Old Dominion Hospital, the Memorial Hospital and the Stuart Circle Hospital—all three of which he was president at various times—owe to him more than to any other individual the success of their existence. In the field of surgery he reached a rank of commanding importance in his section of the country and was widely known throughout the remainder of it. Though retaining to the end his grasp upon the general field, he devoted the best thought of his latter years to the genito-urinary branch, and it was in this department that he won his greatest reputation. He was identified with many organizations, among them the American Surgical Association, the American College of Surgeons, the American Association of Obstetricians and Gynecologists, the American Urological Association and the Southern Surgical Association. He served as president of the last-named organization and also of the Richmond Academy of Medicine and Surgery.

Though he contributed to standard text books and journals, it was a source of much regret to his admirers that the exactions of a busy life and the restraint of his modesty prevented Dr. Bosher appearing in print more frequently. A thorough student, a close observer, a skilled and careful workman, his judgment ripened into wisdom and his knowledge expanded to encyclopedic dimensions. Only those close to him knew and felt the full breadth of his surgical mind, and to them in particular its passing without more adequate transmission to posterity is a matter of no ordinary concern.

The estimate of his fellow-men, among whom he walked, with whom he worked, and upon whom he practised, is the surest approach to the character of Dr. Lewis Crenshaw Bosher. The Richmond profession, regardless of affiliations, gave him admiration, confidence, and affection. The community in which he resided had long recognized him as one of its first citizens. His patients were forever afterward his friends and apostles. To those privileged to know the depths of his great heart he was in all truth and sincerity a knight, "Sans peur et sans reproche." S. Mc.G.

DR. HARRY M. SHERMAN

"The work that I was born to do is done."

THESE words of George Chapman very aptly summarize the purpose, the devotion and the complete resignation of the friend, the colleague, the teacher, the loving husband and the father, who in passing left a rich legacy—the memory of a life full of service!

As the end, of which he was fully conscious, approached there was no abatement of his faith or his enthusiasm. Weary with incessant effort—a sufferer from an affliction that for years had been his nemesis—he labored on without murmur of complaint. Work seemed to be his only respite—the relief of suffering and the thought of his loved ones his chief incentive.

As an after-word, he might well have said with Chapman:

"For me, let just men judge by what I show
In acts exposed how much I err or know."

Dr. Harry M. Sherman, 1854-1921, was born in Providence, R. I. He attended St. Paul's School at Concord, N. H., and was graduated at Trinity College, Connnecticut. He received his medical degree at Columbia University (then the College of Physicians and Surgeons), New York, in 1880. He served his internship at Bellevue Hospital, and after a brief interval he went to San Francisco, Calif., in 1885 and engaged in practice. Rapidly he rose to a position of leadership in his state and nationally. His prerogative as a molder of opinion and conduct was universally recognized and rarely disputed throughout an honorable career.

His was a strong, manly, robust influence in all activities, civic and professional, that spoke eloquently for the uplift and the welfare of mankind.

In the campaigns waged locally against bubonic plague and tuberculosis, he was a tireless, efficient worker; and later as regional director of the American Society for the Control of Cancer he inaugurated a movement which exacted a large measure of his time and drew heavily upon his physical resources.

His larger scientific and professional interests led him very early into the field of orthopedic surgery. After prolonged, painstaking preparation abroad he returned to California as a pioneer in this department and was at once made associate in surgery, in charge of orthopedics, in the University of California. Through his activities

as a teacher, author and original worker he became a recognized authority in his specialty. For thirty-five years he served continuously as a member of the staff of the San Francisco Children's Hospital, where he did his noblest work; here he blazed a trail that has become a beaten path for future generations.

In 1899 he was made professor of surgery in the University of California. He assumed this responsibility with many misgivings, and only after an earnest protest. But the type of man he represented was an urgent need of the hour, and at great personal sacrifice he served with distinction in this capacity until 1912.

The part played by Dr. Sherman in the establishment of the present University Hospital is known to but a few of his intimates: The undertaking had its genesis a few weeks after the great fire of 1906. Grave doubts were expressed as to the possibility of its completion, and those most in its interest were confronted by obstacles that seemed well-nigh insuperable. It was due largely to Dr. Sherman's boundless faith that the hospital was made ready and opened April 18, 1907, the first anniversary of the fire, with one bed and one patient. It should be added that the initial equipment and the patient were provided by Dr. Sherman.

The splendid institution which has risen in place of this primitive foundation stands as a monument to the laborious efforts of a small group of men, among whom Dr. Sherman was conspicuous.

For several years Dr. Sherman had been a member of the U. S. M. R. C. When this country entered the World War he engaged actively in the work of the Special Medical Examining Board, where his comprehensive training rendered his service invaluable.

In his sixty-fifth year, because of his special qualifications, he was called to the colors as Major of Volunteers in 1918 and placed in charge of Fort Rosecrans Hospital, San Diego, Calif., where he remained until the signing of the Armistice.

In conclusion, be it said, his was a life of self-sacrifice. Few men have lived more for his fellows and less for self. T. W. H.

DR. OSCAR HUNTINGTON ALLIS

Dr. Oscar Huntington Allis died at his home in Philadelphia May 16, 1921. He was born in Holly, New York, in 1843, and was graduated from Lafayette College in 1864 and from the Jefferson Medical College in 1866, and served later as resident physician at the Philadelphia Hospital. After completing his service at the

Philadelphia Hospital he was connected with the surgical service of the Jefferson Medical College Hospital, and was elected professor of orthopedic surgery in 1882.

He was elected surgeon to the Presbyterian Hospital in 1872, being one of the original members of the surgical staff of that institution. He held this position until he resigned in 1910, when he was elected consulting surgeon. He also served as surgeon to the Howard Hospital. He was elected a member of the American Surgical Association in 1890. He delivered the Mütter Lectures at the College of Physicians in Philadelphia in 1888 and 1916 and also the Lane Lecture at the Cooper Medical College, San Francisco, Calif., in 1903.

He was a frequent contributor to surgical literature. His papers upon the mechanism and treatment of fractures and dislocations are well known to the surgical profession. One of the most notable of his papers was that upon "Dislocations of the Hip-joint," which was awarded the Samuel D. Gross prize by the Academy of Surgery of Philadelphia. His studies in lateral curvature of the spine were very valuable. Dr. Allis was an industrious worker in surgical problems and devoted much time to the study of human anatomy, spending many hours in the dissecting room even during the later years of his life.

He possessed rare mechanical ingenuity, devising and perfecting a number of surgical instruments, which are in daily use by many surgeons at the present time. H. R. W.

DR. CHARLES BEYLARD GUERARD DE NANCRÈDE

Dr. Charles Beylard Guerard de Nancrède was born in Philadelphia, Pa., on December 20, 1847, and died April 12, 1921, at Ann Arbor, Mich. His pre-professional education was received at private schools and at the University of Pennsylvania. As a private student, and later as assistant to the professor of physiology at the University of Pennsylvania and assistant to the professor of physiology in the Philadelphia Dental College, he began his study of medicine. In 1883 he graduated in medicine from the Jefferson Medical College, Philadelphia. The following honorary degrees were conferred upon him: An honorary M.A. by the University of Michigan in 1893 and by the University of Pennsylvania in 1894; the degree of LL.D. by the Jefferson Medical College in 1898 and by the University of Michigan in 1920.

Dr. de Nancrède taught anatomy as assistant demonstrator for thirteen years at the University of Pennsylvania and subsequently was a lecturer on regional anatomy and lecturer and demonstrator of osteosyndesmology in the same school. He was appointed clinical lecturer on rectal and general surgery in the Jefferson Medical College, and served as chief of clinic of one of the eye and ear services at the University of Pennsylvania under Dr. George Strawbridge in 1871-72. From 1900-1913 he held the chair of surgery and clinical surgery at Dartmouth Medical College, giving his course during the summer.

The distinction of being the first surgeon in Philadelphia to operate for bullet-wounds of the stomach and intestines belongs to Dr. de Nancrède. He was among the first to operate for appendicitis, and the diagnosis and surgical treatment of brain abcesses and cortical epilepsies was made a specialty by him. He was an early and persistent advocate of Listerism, and, with Drs. Keen and Mears, has been credited as chiefly responsible for establishing this practice in Philadelphia and the United States. The active and varied experiences of Dr. de Nancrède resulted in his being called to the chair of surgery in the University of Michigan, where he served many years with untiring effort and with splendid results.

His activities were diverse and numerous. Whether as President of the American Surgical Association and the Washtenaw County Medical Society of Michigan, as corresponding member of the Royal Academy of Medicine of Rome, Italy, as recorder and editor of the *Proceedings of the Pathological Society of Philadelphia* or member of the many other societies to which he belonged, including the American Medical Association, the International Society of Surgery, the Philadelphia Academy of the Natural Sciences and the Northern Tri-State Medical Society, he could always be counted upon to take an interested, instructive and valued part.

His contributions to medical literature were most voluminous. He was the author of important monographs on the *Principles of Surgery* and the *Essentials of Anatomy*, and was a valued contributor to various systems of surgery: *Internal Encyclopedia of Surgery*, *Cyclopedia of Diseases of Children*, *American Test-book of Surgery*, *Dennis's System of Surgery*, *Park's System of Surgery*, *Treatise on Surgery by American Authors* and *American Practice of Surgery*. His shorter contributions covered almost every field of surgery and gave evidence of the tireless, painstaking energy with which he approached any subject in which he was interested.

He had marked artistic ability, was fond of sketching and drawing, and was a member of the Philadelphia Sketching Club. His artistic

temperament found an outlet in music also, and he became a member of the Philadelphia Choral Society, and later, for years, he sang in the chorus at the University of Michigan.

To his other attributes was added a patriotic and chivalrous devotion to his country, and each call to service found him among the first to answer. During the Spanish War he served as Major and Surgeon with the Second Army Corps, and he was recommended for brevet Lieutenant-Colonel because of services to the wounded under fire during the Santiago campaign.

As a member of this Association we recall him vividly. Keen and critical in his analysis, positive because of his broad knowledge and reading, a surgical teacher whose contributions were always received with interest and close attention, he was one of our distinguished elder leaders, and the Association sincerely mourns his loss.

F. E. B.

DR. FREDERIC HENRY GERRISH

Dr. Frederic Henry Gerrish, son of Oliver Gerrish and Sarah Little, was born March 21, 1845, and died September 8, 1920, at Portland, Me.

He was educated in the public schools of Portland, entered Bowdoin College with a high examination rank and was graduated with Phi Beta Kappa honors in the class of 1866.

He received the degree of M.D. from Bowdoin College in 1869, the degree of LL.D. in 1905 from Bowdoin College and also in 1904 from the University of Michigan.

Dr. Gerrish was professor of materia medica at the Bowdoin Medical School for several years, lecturer in public health, professor of anatomy, professor of surgery and professor of medical ethics, and up to the time of his death one of the overseers of Bowdoin College.

Dr. Gerrish was an anatomist, a skilled surgeon, an inspiring lecturer in surgery, a famous teacher, a sympathetic physician and a clear writer on surgical subjects. He wrote *A Text-book of Anatomy*, published in 1899, which has been recognized as a standard work. He translated the book of Lucas Championnière, *Aseptic Surgery*.

Dr. Gerrish was most active in introducing aseptic surgery into surgical practice; he was greatly interested and very active in legislation on matters of public health and was influential in raising the standard in the education and training of nurses.

He was at one time president of the American Academy of Medicine and of the American Therapeutic Society.

Dr. Gerrish was admired and esteemed by his students, intimately associated with his colleagues and thoroughly trusted and loved by his patients. He contributed notably to the establishment of that high standard of surgery which has obtained in New England.

Mrs. Gerrish was a constant companion and of great assistance to him throughout his long and varied professional life. C. L. S.

MAJOR-GENERAL WILLIAM CRAWFORD GORGAS, M.D.

Major-General William Crawford Gorgas, M.D., was born in Mobile, Alabama, October 3, 1854 and died July 4, 1920. He was stricken with apoplexy in London, May 30, 1920. He had returned from Belgium, where he had been decorated by King Albert, and was journeying to Africa under the auspices of the British Government to investigate cases of yellow fever existing there.

Thus while receiving grateful acknowledgement and honor for what he had done, he was still pressing forward in his humanitarian effort to eradicate yellow fever from the earth.

The world lost a benefactor and the American Surgical Association an honored member. His life illustrated the best phases of humanitarian achievement based on scientific knowledge.

After graduation from the Southern University and the Bellevue Medical school, and after the completion of his internship at Bellevue Hospital, General Gorgas entered the army in 1880 and was soon deeply engaged in the problems of disease control. His attitude during the early years of his army life is illustrated by the fact that while on duty at Fort Brown, Texas, he suffered arrest at the hands of General Crowder for doing an autopsy on the body of a patient dead of yellow fever, although it had been ordered that no officer should come in unnecessary contact with a case of this disease. While at the post he contracted the disease himself and thus acquired an immunity which was helpful to him at a later time.

The Spanish-American War was not encouraging for sanitarians. Although typhoid fever was fairly well understood at that time its control was hardly more efficient than in the Civil War. Yellow fever was less understood, and even General Gorgas had recommended the burning of the village of Siboney, Cuba, in 1898, in order to stamp out the disease. He afterward "wondered how many infected mosquitos were destroyed in that conflagration."

In 1900 the late Major Walter Reed was detailed as head of an army board for the study of yellow fever in Cuba and made his epoch-

marking discovery of the relationship of mosquitos to that disease. General Gorgas was then chief sanitary officer of Havana and immediately made practical use of Reed's discovery.

In 1901 he screened the yellow fever patients and oiled the breeding-places of the mosquitos, and in less than three months Havana was free from yellow fever for the first time in 150 years.

In 1904 he was appointed chief sanitary officer of the Canal Zone. The French had lost 22,819 laborers during their nine years' work in this region. When General Gorgas began his work there the death-rate was as high as ever—a yellow fever epidemic was actually in progress. In less than a year the disease was completely eradicated, and has not since recurred. Under General Gorgas's administration and the continuation of his methods the Canal Zone has become one of the most healthful parts of the world.

General Gorgas maintained charge of the sanitation there until 1913, when, at the invitation of the Chamber of Mines of Johannesburg, he went to South Africa to investigate the high death-rate among the miners at the Rand. By the application of the fundamental principles of hygiene he lowered the death-rate and improved the condition of the miners.

In 1914 he was appointed Surgeon-General of the United States Army. It is fortunate that he was in charge of the army's health during the war. He was familiar with the intricate details of the situation; he had the respect and affection of his co-workers; and he was ever mindful of the enlisted men. Under his direction the "mobilization of a great army was accomplished with a lower death-rate than had hitherto been known in the annals of military medicine" (Victor C. Vaughan).

During a part of his term as Surgeon-General and after the conclusion of that term he investigated the localities of epidemic yellow fever in South America. He hoped to eradicate the disease in each of them.

He received many honors during his career. The United States Government made him Colonel and Assistant Surgeon-general by a special act of Congress on March 9, 1903. In 1914 he was appointed Surgeon-General of the United States Army with the rank of Brigadier-General, and in 1915 he was made Major-General. He received degrees from many American universities and the degree of Doctor of Science from the University of Oxford. He was decorated by King Albert of Belgium and was made a Knight Commander of St. Michael and St. George by the King of England. He was awarded gold medals by the Liverpool School of Tropical Medicine and by

the American Museum of Safety. He was elected to honorary membership in our own Association in 1916.

During all his career he showed qualities of heart and mind which we all admired and loved. He was large-hearted, sympathetic, considerate, kindly. The rank and file of the soldiers especially appealed to him; he was in close touch with them, and ever was the guardian of their physical welfare. His achievements were stupendous, and easily placed him among the great benefactors of mankind.

In 1885 he married Miss Marie C. Doughty, of Cincinnati, Ohio. She and a daughter, Mrs. William D. Wrightson, survive him.

C. N. D.

ADDRESS OF THE PRESIDENT

THE MAKING OF A SURGEON

By JOHN B. ROBERTS, M.D.
PHILADELPHIA

Education in the medical specialties has been given a great deal of attention by our profession within recent years. Surgery has been developed to such a degree of accuracy that the training of those fitted to practice its various subspecialties satisfactorily, honestly and safely has become of paramount importance. The mental and moral equipment of a medical graduate seeking professional and public support in his operative surgical efforts should be subjected to a searching analysis.

This organization of teachers and research workers in surgery is one of the agencies which may well devote time to the consideration of "The Making of a Surgeon." The President therefore has grouped observations of some early writers on surgical teaching from the time of Paracelsus down to the present century.

When Paracelsus at the University of Basel, in Switzerland, insisted that he would teach not in the Latin of the scholastics but in the language of his hearers, and laid stress on personal observations and research in surgical education, he opened the way to modern surgery. Arderne and Gale, in England, subsequently prepared text-books in English for the training of anatomists and operators. Later, John Hunter and his pupils developed the surgical science and art in England, from which our particular Fellowship has largely drawn inspiration and skill.

This line of study soon brought me to the lives and work of men with whom we came into personal contact who now guide, to a considerable extent, the surgical thought of our respective medical schools and communities.

A review of the methods of study which developed Samuel David Gross, the founder of the American Surgical Association, William Williams Keen, a survivor of the original active Fellows, and a glance at the life-work of its Associate Fellows, Marion Sims, Joseph Lister, Theodore Kocher, Victor Horsley and William C. Gorgas, give evidence of what is needed to make surgical science have a spirit of efficiency, mercy and altruism. These men have transmitted to this assembly of my colleagues a glorious heritage.

A conviction has been brought to me, by these studies, that a maximum of success up to the limit of an individual's native capacity may be best obtained by certain educational equipments acquired prior to the entrance into the undergraduate medical school and after release from the now usual hospital internship.

A real surgeon should have knowledge of logic, psychology, ethics, and at least a familiarity with the fundamental processes of rather advanced mathematics and of chemistry and physics. Otherwise, how can he be expected to accurately use his reason to understand and interpret the mysteries of biochemistry, physiology, and the physical processes of life?

Similarly, he needs, particularly in a surgical career, a working knowledge of general mechanics and should have some training of his hands and fingers, by which his cerebral centers will be stimulated to specific reactions.

In addition to an acquaintance with Latin, he should know some Greek. These tongues lie at the basis of medical nomenclature.

With this equipment, let him enter the medical school wherein, among other topics, medical history should not be neglected.

The hospital internship, now so wisely insisted upon in some states, should be followed by, at least, one or two years' general

practice. This should, if possible, be under the partial supervision of an internist of ability, conscientious temperament and endowed with a practical knowledge of how to care for distempered souls as well as impaired bodily organs.

The youth is now ready to begin a preparatory course for a true chirurgical life—the happiest, the best and the most glorious of man's efforts for his fellow-men. In this graduate study he must return to first principles and renew acquaintance with anatomy, morbid anatomy, pathology, physics and chemistry with eyes, ears and a brain of an intellectually receptive mind. The sciences allied to medicine will be part of his systematic study.

He should later make application of his basic education in science and medicine and his renewal of scholastic topics to the diagnosis and treatment—operative, medical and physico-therapeutic—of surgical conditions under wise guidance; and, finally, by research work in connection with his operative opportunities, take place among the recognized surgeons of his country.

THE SURGICAL TREATMENT OF MEGACOLON

By CHARLES N. DOWD, M.D.
NEW YORK

It is nearly a century since Parry described a case of megacolon, observed at autopsy in an adult who had suffered from digestive disturbance for years. This colon was enormously distended and contained an immense quantity of feces.

We do not know what observations had preceded his, but between 1825, the date of his publication, and 1886, the date of Hirschsprung's first article, there were at least twenty-six publications about these tremendously enlarged colons.

Hirschsprung's articles stimulated attention to the subject, and they came at a time when abdominal surgery and abdominal pathology were receiving increased attention, hence when Finney wrote his classical article in 1908 he found a bibliography of 206 articles.

Since that time the literature has increased even more rapidly, and is now so large that it is hardly possible to grasp it in full detail.

Method of Occurrence. Congenital idiopathic dilatation of the colon (Hirschsprung's disease) has generally been considered, in large degree, a disease of childhood. There are a great many descriptions of the disease occurring in children. They surely make the most vivid pictures. Hirschsprung advocated the name of "true" megacolon for them and the name of "pseudo" megacolon in those cases occurring in adults. With the later development of the subject this distinction has not always held. Many observers believe that if some patients with this disease can worry along to adolescence others can

reach adult life, and that there is no proper age limit. The subject has been complicated by the including of reports of localized megacolon. Even in the undoubted congenital cases the sigmoid is more liable to show dilatation than the rest of the colon. The intestine angulates at the fixed points and distends elsewhere, although after its removal it may be as large at a point of former angulation as at a point of former dilatation. Hence it is not strange that there should be many reports of localized megacolon. However, the custom of reporting such cases has led to the description of an increased number of adult cases and an increased proportion of cases in which the mechanical factor of obstruction predominates. Considerable confusion has resulted, and it is now impossible to differentiate in the literature those patients in whom the enlargement of the colon is due to a congenital defect from those in whom it comes from mechanical or spasmodic obstruction. We know, however, that enormously dilated and hypertrophied colons are observed in children and that similar conditions are sometimes seen in adults; also, that the adult cases in the main show greater evidence of mechanical or nervous obstruction as causative factors.

Pathology. The degree of dilatation which these colons reach is almost beyond belief.

Finney states that the circumference of the abdomen may be greater than the height of the patient. Graves describes a colon 110 cm. long and 31 cm. in circumference in its descending portion. Aronson describes one eight inches in diameter; Bensandi and Sorrel, 26 cm. in circumference; Jackson, 18 cm. in circumference. Johnson describes a colon which contained three or four quarts of feces. From the second case recorded in this paper at least one gallon of semifluid feces was withdrawn on the operating table and about one gallon drained away in the next two days. The removed portion of the colon in Case I easily held four liters of fluid after its removal. Holt quotes Hughes as describing in a boy, aged three years, a colon which held fourteen pints of water, and Finney quotes Peacock as describing one which contained sixteen liters.

The walls of the colon are usually hypertrophied as well as dilated, especially in their circular coats.

The mesocolon may be lengthened and hypertrophied in all its elements.

Obstructive valves have been described.

Volvuli have been found.

A few anal fissures have been described giving suggestion of spasmodic obstruction above.

Etiology. There are three main theories for the etiology:

1. That it is due to congenital defect in the development of the colon.

2. That it is due to some form of mechanical obstruction and that the colon hypertrophies and dilates in its efforts to overcome this obstruction, just as the heart hypertrophies and dilates when increased demand is made upon it.

3. That it is due to spasm of the circular fibers in the lower part of the intestine, and that dilatation of the colon follows this spasm just as dilatation of the esophagus follows cardiospasm or dilatation of the stomach follows pylorospasm.

It is altogether probable that two or more of these elements are active in many of the cases.

Symptoms. The main symptoms are abdominal distention, constipation lasting sometimes for very long periods, intermittent diarrhea, loss of appetite, emaciation and apathy.

The study of the treatment of the disease is the main purpose of this paper. Very careful consideration of its occurrence, pathology, etiology and symptoms is given in other papers, particularly that of Finney, to which readers are referred for further information.

Record of Cases

Case I.—E. F., aged ten and a half years. Roosevelt Hospital. History No. 16873 A.

The patient was first seen by the writer March 31, 1920. She had reasonably good health during the first seven years

of her life, then had loss of strength and listlessness. At eight and a half years irregular attacks of abdominal pain began in the epigastrium and on both sides. They were accompanied by constipation. Her disability progressed. By December, 1919, she had become a real invalid, taken about from one doctor to another in search of relief from her distress, weakness, pain, alternating constipation, diarrhea and frequent micturition.

When admitted to the hospital she was found to be a thin emaciated child of sixty-one pounds weight and having a very large abdomen. The left half of the abdomen contained a large, rather soft, slightly movable mass. Enemas brought away an enormous quantity of feces and the mass disappeared. No obstruction could be found in the rectum. X-ray examination after a barium enema showed a dilated sigmoid (Fig. 2). It was not found practicable to give enough barium mixture either by the mouth or the rectum to secure a picture of the entire colon in a dilated condition.

Operation. After ten days of further treatment by medicine, diet and enemas an operation was done. A seven-inch incision was made through the inner part of the left rectus muscle; a greatly distended transverse and descending colon were immediately encountered. They seemed to fill the greater part of the abdomen and to push the rest of the intestines backward and to the right. The splenic flexure was held in position by its peritoneal attachment, the costo-colic ligament, and formed an acute angulation. The distention seemed largely due to gas.

The descending colon and the splenic flexure were mobilized by dividing their outer leaf of investing peritoneum. The mesocolon was then divided in sections from the lower sigmoid to the hepatic flexure. The liberated loop of intestine, somewhat over forty inches in length and four inches or more in diameter, was then drawn outside of the abdomen through an intramuscular incision which had been made for that purpose above the left anterior superior iliac spine. This brought the hepatic flexure into contact with the lower sigmoid without undue tension. The afferent and efferent intestinal walls were

stitched together within the abdomen to aid in proper "spur" formation.

FIG. 1.—Diagrammatic drawing showing condition of megacolon in Case I. Colon angulated at splenic flexure. Transverse colon and descending colon and sigmoid flexure greatly distended.

The median wound was then closed and sealed. The intestine at the place of exit was secured to the abdominal wall. Tempo-

rary circular ligatures were applied to it outside of this exit and the loop of colon beyond the ligatures was ablated. The ablated portion was 95 cm. long and held four liters of fluid when injected.

She showed considerable shock and reaction after her operation, but finally rallied satisfactorily. The temporary circular ligatures were removed from the protruding stumps of intestine after forty-eight hours and a "double-barreled" intestinal stoma was thus established.

FIG. 2.—*X*-ray picture after an enema of barium mixture, showing shadow of the descending colon and sigmoid flexure.

The median incision healed by primary union. The clamping of the spur was begun after twenty-five days, and twenty-three days later the intestinal ends were stitched together at the site of the stoma.

She left the hospital seventy-eight days after the operation. At that time the stoma was firmly closed. She was having normal bowel movements; she had been walking and playing about the ward and the porch for two or three weeks; she ate

well, had gained much strength and five and three-quarter pounds in weight. The *x*-ray picture shown in Fig. 3 indicates the condition of the remaining colon at that time.

She has been carefully followed during the intervening months. Her digestion has remained normal and she has normal daily stools. She has gained twelve pounds in weight since she left the hospital and is now a normal, active, happy, good-natured youngster.

FIG. 3.—*X*-ray picture taken twenty-four hours after a meal of barium mixture, showing condition of colon three months after operation.

An *x*-ray picture taken March 7, 1921, shows that the colon has about the same outline as when she left the hospital.

Pathological Report. Macroscopic. The specimen consists of a piece of large intestine 95 cm. long and in transverse diameter, varying from 5 to 7 cm. The wall is thin, markedly dilated, pale and of atonic appearance. The mucosa appears normal and the specimen otherwise is unremarkable.

Microscopic. The entire wall is very much thinned out. The mucosa is intact and shows no inflammatory reaction. The

muscularis mucosæ is very thin. The submucosa consists of relatively loose tissue. The circular and longitudinal muscles are thinned out. The serosa is loose and shows no connective-tissue increase. The layers are about one-half the normal thickness.

Diagnosis: Megacolon.

W. C. White, M.D.

Case II.—F. W., aged seventeen years. Admitted to the Roosevelt Hospital November 6, 1920. History No. 17337 A.

She had always been very constipated; has had three or four attacks of great abdominal distention and vomiting during her life. The last previous attack came six months ago; the distention then was almost as great as at the present time. She then had repeated vomiting and severe abdominal "cramps."

The present attack began three weeks ago with abdominal distention, nausea and vomiting. The distention increased and was accompanied by very severe abdominal pains. Repeated enemas had been given, but some of them at least did not return. The bowels, however, had moved two or three times a day much of the time. During the past three days she had many times vomited green offensive material. She had been in bed the entire three weeks and had become very weak.

On admission to the hospital she was emaciated, very feeble, pulse 140 and the abdomen was enormously distended. The outlines of the intestine were visible through the thin abdominal wall, and slowly moving peristaltic waves could be seen. Enemas and rectal tubes were used in an effort to empty the bowel, but they were not successful. Operation was therefore done.

Through an incision in the lower part of the left rectus muscle an enormously distended sigmoid flexure was found. It was about as tense and thick-walled as an ordinary large ovarian cyst. It could be traced from the pelvis to the upper left angle of the abdomen, but satisfactory examination of the colon beyond that point was not practicable. A suction trocar withdrew

semisolid feces. A purse-string suture was placed about the trocar wound. The abdominal incision was closed except an opening of one and a quarter inches; the intestine outside the purse-string was stitched to the edges of this opening; a flanged glass tube was introduced within the purse-string and secured in its position. About one gallon of fecal material was then drawn through the tube by a suction apparatus. During the following forty-eight hours nearly another gallon of fecal material drained away. The abdomen became flat and its circumference was reduced from thirty-four inches to twenty-six and a half inches.

For a few days she showed some recuperative power, but died on the sixth day after her operation, apparently with a low grade of pneumonia. She showed no signs of peritonitis. Autopsy was not allowed.

Case III.—J. C., aged forty-two years. Admitted to the first surgical division of the Roosevelt Hospital April 21, 1921.

He had suffered from various forms of digestive disturbance during much of his life. Appendicectomy was done in 1909. Gastroenterostomy was done in 1912. He had then enjoyed reasonably good health until two weeks before admission to the hospital, when he had an attack of bronchitis or broncho-pneumonia.

Sixty hours before admission he was seized with very severe general abdominal pain—he was "doubled up with the cramps." Vomited repeatedly—attacks of vomiting and pain have both continued until the present time. Constipation "very bad" for the past two weeks. Bowels did not move without catharsis, and then only in small quantities. Much worse since pain started. No gas per rectum.

When admitted to the hospital he seemed weak, thin, very ill and in much pain. His abdomen was distended and showed much muscle spasm, with dulness at its lower part. Peristalsis waves were visible.

Operation. A four-inch incision was made in the right rectus and was enlarged to six or seven inches. Abdominal contents under much tension. The small intestine was collapsed and was apparently empty throughout its entire extent.

In the left side of the abdomen, extending from the diaphragm into the pelvis, there was an enormously dilated portion of the large intestine. In order to deliver this it was necessary to remove much of the small intestine from the abdomen. It was temporarily confined in warm, moist towels. The distended colon was then found to extend from the ileocecal valve to the beginning of the transverse colon. It was delivered with considerable difficulty. The large intestine below the middle of the transverse colon was not dilated. The constriction seemed to have been caused by the hinging of the gut at the middle of the transverse colon. The entire ascending colon had been transferred to the left side and had become enormously dilated in that position.

An effort was made to push the gas downward from the dilated intestine. This was only partially successful, although no definite band of constriction could be found. A rectal tube was inserted through the anus to aid this procedure.

The outer layers of the dilated intestine were dark in color and the wall had broken down in several places. It was therefore deemed wiser to leave it outside the abdomen than to make further efforts to return it. The wound was therefore closed in layers to the emergence of the intestine.

The distended intestine was then punctured with an aspirating trocar and a large amount of gas withdrawn, also some very foul-smelling fecal material.

The afferent and efferent legs of the intestine were sewed together. The intestinal wall was stitched to the skin. The wound was smeared with sterile vaselin. The ascending colon, which was still somewhat dilated, was left in the dressing and the patient returned to bed in this condition.

After two days a part of the protruding intestine was excised and sent to the pathological laboratory.

Pathologic Report. Macroscopic. Specimen consists of an opened and somewhat dilated cecum preserved in formalin. It is more or less cylindric in shape and measures 13 cm. in the longitudinal axis and varies from 2.5 to 6.5 cm. in transverse diameter across the opened surface. The serosa is rough and has some adhesions. The wall everywhere is thickened, measuring in many places 1.4 cm. It is firm and fibrous. The mucosa is smooth and thrown into folds. There are no areas of ulceration, necrosis or hemorrhage. The lumen is patent and varies from 2 to 5.5 cm. in transverse diameter.

Microscopic. The mucosa is about normal in thickness, but the submucosa is greatly increased with newly formed connective tissue. The submucosa is vascular and has a round-cell infiltration throughout. The circular muscle is about three times as thick as that shown in Case I, but the longitudinal is about the same in thickness. The serosa is greatly thickened and shows a chronic inflammatory reaction.

Wm. C. White, M.D.

He did well; the wound healed firmly to the emergence of the intestine. Clamps were applied to the spur. On May 31 the ends of the intestine were sewed together.

At the present time he is still under treatment in the hospital, the stoma not yet being closed.

[He left the hospital in good condition with stoma entirely healed July 20, 1921.]

These three cases illustrate various phases of the problem. One of them shows the good result which may come from the removal of a sufficient portion of the enlarged colon. One shows that a patient with this disease may drift along beyond the possibility of relief, either medical or surgical, and that this drifting may occur while she is trying to attend to the ordinary duties of life. Two of the cases show the comparative safety of the extraperitoneal two-stage method of resection (Mikulicz). The cases, however, are too few for general deductions. They

are only to be considered in conjunction with the extensive literature which now exists.

Consideration of the Literature. As a starting-point it should be stated that this literature gives abundant proof that surgical treatment is more successful than medical treatment. For instance, Duval gives 74 per cent mortality from medical treatment and 34 per cent from surgical treatment. Lowenstein gives 66 per cent from medical treatment, and 48 per cent from surgical treatment. Ladd has given the following tabulation of 118 cases:

Treated	Total.	Cured, per cent.	Improved, per cent.	Unimproved, per cent.	Died, per cent.
Medically . .	60	12	13	6	67
Surgically . .	58	41	8	3	41

Terry found a mortality of only 27 per cent in 95 cases treated by surgery. Neugebauer, quoted by Hubbard, referred to 123 cases treated medically with only 1.5 per cent cure.

Surgical Treatment. The term surgical treatment is a very elastic one and includes many forms of procedure. It is interesting to know how different surgeons have met the problems of this disease, and it is also important to know the results of the different forms of treatment which they have used.

A number of tabulations have been made, but we know of none which give the statistics of a large number of cases treated by modern surgery. Hence, with the aid of Dr. Charles W. Lester, the literature has been searched for records of operations since 1908, the time of the publication of Finney's article. The search has been carefully made, and without doubt most of the published reports have been found. Some have probably escaped notice, but no report which has been found has been omitted. 143 cases have been tabulated. This includes 136 cases found in the literature, 4 cases learned of by personal report and the 3 cases recorded in this paper.

Table I shows an improvement in the general mortality rate. So far as we know 29 per cent is a lower mortality rate than has previously been reported for a large collection of cases, excepting Terry's report of 27 per cent.

TABLE I.

	Total.	Cured.	Improved.	Unimproved.	Died.	Mortality rate.
One-stage resection	42	28	3	...	11	26%
Two-stage resection, intra-abdominal method	31	23	2	1	5	16%
Two-stage resection, extra-abdominal method (Mikulicz)	13	11	1	...	1	8%
Colostomy, cecostomy and appendicostomy	20	3	4	...	13	59%
Short-circuiting	22	13	...	2	7	32%
Colon plication	2*	2				
Colopexy	3*	2	1			
Colon plication and colopexy	1*	...	1			
"Laparotomy"	1	...	...	...	1	
Laparotomy with massage of the colon	2	...	...	...	2	
Incision of stricture	1	...	...	1		
Release of volvulus	4*	3	...	...	1	
Colotomy	1	...	1			
Total	143	85	13	4	41	28.7%

The mortality rates shown in some previous tabulations are here given:

	Date.	Number of cases.	Mortality rate, per cent.
Lowenstein	1907	44	48
Schmidt	1908	71	48
Duval	1910	..	34
Schneiderholm	1915	143	36
Ladd	1908	58	41

The improvement seems to be due to the more general use of rational radical surgical procedures. The increase of such surgical procedure is strikingly shown in comparing the table given above with that prepared by Schmidt in 1908. (See Addenda.) It is noteworthy that although Schmidt's tabulation contained

* Other patients who had secondary operations when these procedures had failed to cure are grouped under "two-stage" resections, hence this tabulation does not show the failures of these semipalliative operations.

only five two-stage resections, intra-abdominal and extra-abdominal, they were all followed by recovery.

From a surgical standpoint these cases may be divided into three groups:

1. Those for whom only colostomy or appendicostomy were done.

2. Those for whom there was only some semipalliative procedure such as short-circuiting, colopexy, relief of volvulus, colon plication, etc.

3. Those for whom more or less of the colon was resected.

GROUP 1. The first group should hardly be considered surgical. If patients drift along into a semimoribund condition, and then apply to a surgeon for relief of obstruction, they show the ill-effects of whatever treatment they have previously had. If the surgeon is magnanimous enough to try to relieve their condition by colostomy he makes a praiseworthy effort to save life, but he has not had a reasonable opportunity of curing megacolon by surgery. It is not strange that there was a 59 per cent mortality among the twenty patients in this group. That mortality, however, cannot fairly be considered surgical. Of course this group does not include those cases in which the colostomy was the first step in a two-stage operation.

GROUP 2. Operations of the type here mentioned were common in the earlier reports. In this tabulation they represented less than 26 per cent of the total. Surgeons now prefer procedures which give more prospect of permanent cure. The mortality rate of this group is 7 per cent higher than for the combined three groups of resections and the records of "cures" are 18 per cent less. Secondary operations were frequently done on patients who had previously had one of these semipalliative, but such cases are recorded under two-stage operations.

GROUP 3. *Partial Colectomies.* At the present time the main surgical interest centers in the problem of removing as much of the enlarged colon as is practicable, and this is the obvious method of procedure. There are three main methods of doing this:

1. In one stage, removing a portion of the colon and uniting the divided ends within the abdomen either by end-to-end, end-to-side or lateral anastomosis.

2. Using two stages for similar resection. There are many variations of the two-stage method; a preliminary colostomy may provide for washing out the colon before the resection and may provide a vent afterward. The cecostomy or appendicostomy may be done for a vent at the time of the resection. A short-circuiting may be done first and a removal of the excluded intestine later. A release of a volvulus or colopexy or colon plication may be done first and a resection later.

3. An extra-abdominal resection may be done. The portion of colon which is to be removed may be mobilized and delivered outside the abdominal wall before it is opened, preferably through a separate incision. After stitching the intestine to the abdominal wall and closing the primary incision completely and the secondary incision to the point of emergence of intestine, the lumens of the gut are temporarily closed and the desired portion of colon ablated.

The dangers of these procedures diminish in the order given. The table gives the mortality rates as 26 per cent (Group 1), 16 per cent (Group 2), and 8 per cent (Group 3), a rate which may vary with the next table made but which corresponds in fair measure with other statistics.

Each surgeon will endeavor to fit the procedure to the individual case. The proportion of cases of real megacolon which are suitable for a one-stage resection must be small. The main difficulty is one of cleansing. No doubt some large colons can be so cleansed as to make one-stage intra-abdominal resection justifiable, but most of such resections will need some provision for possible leakage at the suture line and for escape of gas from another part of the intestine.

If operation in two stages is decided upon, each surgeon will again endeavor to fit the procedure to the individual case. Extraperitoneal resection (Mikulicz) is often more tedious and trying to patient, nurse and surgeon than other forms of two-stage operations.

It has never been generally popular, as is shown by the limited number of reported cases. It is, however, becoming more popular and is a very valuable resource; it is doubtful, for instance, if Case I would have survived and obtained so good a result under any other form of operation. It has the great advantages of rapidity, diminished manipulation of the intestine and diminished shock to the patient. It gives less likelihood of troublesome adhesions. The danger of infection is very greatly diminished, since the portion of intestine which is to be ablated is delivered outside the abdomen before it is opened.

It is quite possible that further development of the two-stage intra-abdominal method may meet these advantages; that the colon may be adequately cleansed; that suitable provision for possible leakage at the suture line may be made and that an intestinal vent may be provided which will prevent the accumulation of gas. Also that the patient's strength may be so conserved as to provide a reasonably safe margin for extensive operation.

It is, however, to be remembered that most of these operations are done under adverse conditions. The patients are weak and debilitated, the procedure is an extensive one, the resources of surgery are put to a maximum test and the procedure which gives the least danger must often be selected even if it implies a more disagreeable convalescence than one would wish. Hence the two-stage extra-abdominal method (Mikulicz) is our safest resource for a larger proportion of these patients.

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ADDENDA

SCHMIDT'S TABULATION OF CASES OF HIRSCHSPRUNG'S DISEASE TREATED BY SURGERY. PREPARED 1908. BEITR. Z. KLIN. CHIR., VOL. LXI, p. 682.

		Cure.	Improved.	Not improved.	Died.
Expl. lap	11	3	2	1	5
Puncture	4	..	..	..	4
Artificial anus	10	1	..	1	8
Colotomy	5	..	..	..	5
Plication	2	1	..	1	
Colopexy	6	4	1	1	
Anastomosis (short-circuit)	16	6	3	..	7
One-stage resection	12	6	..	1	5
Two-stage resection	4	4			
Extra-abdominal resection	1	1			
Total	71	26	6	5	34 = 48%

AGE OF PATIENTS RECORDED IN TABLE I.

Age.	Total.	Cured.	Per cent.	Improved.	Per cent.	Not improved.	Per cent.	Died.	Per cent.
Under 6	27	10	40	3	12	..	..	12	48
6–10	17	10	59	..	..	2	12	5	29
11–20	21	16	76	..	..	1	5	4	19
21–40	37	27	75	3	8	1	3	5	14
41–60	22	15	68	..	..	..	..	7	32
Over 60	11	4	36	1	9	..	..	6	55
Not stated	8	7	87	..	..	..	..	1	13

CAUSE OF DEATH OF PATIENTS RECORDED IN TABLE I

Peritonitis	16
Shock and collapse	13
Embolus	4
Pneumonia	2
Hemorrhage	1
Other causes	5

CARCINOMA OF THE LOWER PART OF THE SIGMOID IN A BOY FOURTEEN YEARS OF AGE

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CARCINOMA of the large bowel in *young* persons is a rare condition. Louart,[1] in 1900, collected from the literature only 9 cases occurring in youths up to twenty years of age. Clott,[2] in 1908, collected 72 cases of cancer of the colon. The youngest of these patients was fifteen years of age.

Merkel,[3] in his article on "Tumors of Childhood," in 1912, states that "the literature on the subject of malignant tumors in childhood must be accepted with some degree of caution, since many of the cases lack histological confirmation."

Weinlechner, in 5279 cases of carcinoma, observed 18 (0.34 per cent) among children up to fourteen years of age. Feldner, in 914 cases of carcinoma, met with only 3 up to the sixteenth year of life.

Philip in his inaugural dissertation on "Carcinoma in Childhood" (Munich, 1908), reviews the older and more recent literature. Among 390 cases collected by him only 87 seemed sufficiently genuine to warrant consideration. Classified according to location and organs involved, he found the following regions to be entirely immune to carcinoma: Lungs, tongue, esophagus, hypophysis (Erdheim, however, subsequently published 2 cases

[1] Thèse de Paris, 1900.
[2] Lancet, 1908, ii, 1007.
[3] Handb. d. allgem. Path. u. path. Anat. d. Kindesalters, Wiesbaden, 1912, Pt. I, vol. i.

of malignant growth of the hypophysis), urinary bladder, penis, seminal vesicles, tubes, mammæ. Merkel calls attention to the fact that the list comprises precisely those organs in which carcinoma in the adult is referable to chronic irritation.

The digestive tract was affected in 46 per cent of the cases, and the sigmoid in only 5.4 per cent. In 111 cases the large bowel was affected in 28; of these, the sigmoid was involved in 7 cases.

Parkinson[1] cites a boy, aged nine years, who died six months after onset of symptoms. No operation. The autopsy showed that the abdominal lump (palpable *intra vitam*) proved to be the sigmoid lying in the right iliac fossa and adherent to the adjacent small intestine. The wall of the sigmoid was thickened with a hard, whitish growth; the inner surface was ulcerated, no trace of mucous membrane remaining. The wall was one-third inch thick, the external surface was smooth. There was no constriction of the gut. Other organs normal. Microscopic sections showed "large rounded and tailed cells packed in the midst of fibrous tissue alveoli. In some parts this is replaced by a granular, structureless, apparently colloid substance taking the stain badly. It is, I think, a carcinoma." The author also refers to a case observed in a girl twelve years of age.

Barber[2] reported a case of a boy aged nineteen years. He was admitted to the hospital with symptoms of acute intestinal obstruction. Cancer history in the family, the mother having died of intestinal carcinoma. The patient gave a history of intestinal cramps for one year, aggravated during the past two weeks, causing inability to work. He had always been constipated, but had no pain when the bowels acted. Two days before entering the hospital the patient vomited persistently. There was no evidence of blood in the vomitus or colonic washings. The abdomen was very tender, rigid and distended, especially on the left side and at the umbilicus. A median incision disclosed a large indurated mass involving the upper

[1] Proc. Roy. Soc. Med., 1913–14, Sect. Dis. of Child., vii, 9.

[2] Interstate Med. Jour., 1919, xxvi, 106.

extremity of the sigmoid. Above this mass the intestine was very much distended. There was very marked enlargement of the lymphatic glands in the mesosigmoid and upward in the pre-

FIG. 1.—Case—Gordon Smith. One inch of the bowel above and below the tumor has been trimmed off in mounting the specimen, which on removal was seven inches in length. The width of the base of tumor is two inches.

aortic lymphatic glands. Complete excision was impossible. The tumor and regional lymph glands were excised. An end-to-end union of the bowel was made. The patient recovered and left the hospital improved. A later report stated that he had

lost weight and strength, which indicated that he was undoubtedly suffering from metastases. The pathological report was adenocarcinoma.

The patient, G. S., aged fourteen years, entered the Hamilton General Hospital on February 16, 1920, complaining of pains

FIG. 2.—Case—Gordon Smith. H. P. field of section of tumor. Magnification × 10.

in the abdomen, frequent stools of blood and mucus, with rectal tenesmus. He gave the following history: About four months ago he began to suffer from irregular abdominal pains, sickness and nausea when taking his meals and frequent desire to go to stool. He lost flesh and the spasmodic abdominal pains became more frequent, and when at stool, instead of passing

fecal matter, only a little blood and mucus came away. This was always accompanied by spasm and pain in the rectum. At first the pains were more frequent during the day, especially so when eating his meals, but during the past few weeks he was compelled to go to the toilet two or three times during the night and the rectal tenesmus was more pronounced.

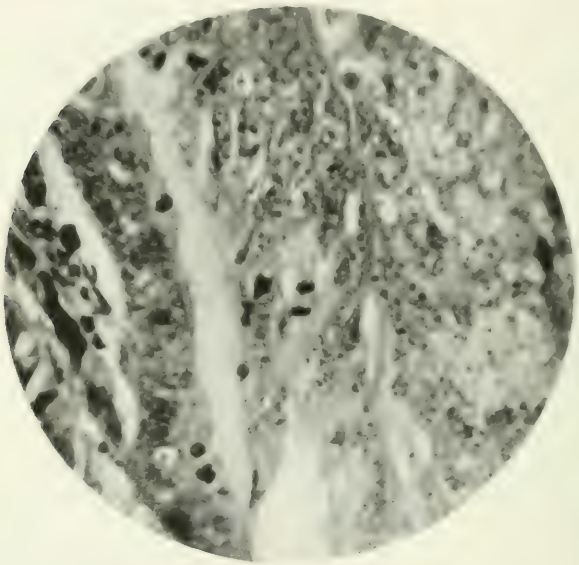

FIG. 3.—Case—Gordon Smith. Magnification × 400. Showing mitotic figures.

His family history is good. Both parents are living and healthy and there is no history of tumors or cancer in his family. With the exception of whooping-cough he has had no previous illness.

Present Condition. He is a small, emaciated-looking, little chap, weighing only sixty-two pounds. His face has a drawn expression, with wrinkles between his eyebrows. His heart and lungs are normal. The urine is negative to casts, albumin and sugar. The abdomen is distended and marked peristalsis is seen and felt in the transverse and descending colon. When this peristalsis comes on the descending colon and sigmoid are

shoved over to and even beyond the median line. After the spasm has passed off a lump can be felt at the lower end of the sigmoid. On rectal examination an irregular mass can be distinctly felt. The abdominal wall is so thin that the distended colon can be easily mapped out. The lad stated that sometimes when sitting on the closet, something seemed to come out of his anus.

It was somewhat difficult to decide the exact nature of the growth, as it hardly seemed likely that a boy so young would be affected with cancer of the large bowel, yet had this condition been seen in an adult fifty years of age I would have had little hesitation in making a diagnosis of cancer. The patient had been in the hospital nearly two days before I saw him and as his condition was not very good I operated immediately.

Operation. The abdomen was opened and the growth was found at the junction of the rectum and lower end of the sigmoid. The bowel above the growth was very much distended with fecal matter. The rectum was likewise very much dilated and there was a partial intussusception of the tumor into the rectum. No enlarged lymph glands were found and no nodules could be felt in the liver.

Owing to the great mobility of the sigmoid and descending colon the mass was easily brought well out of the abdominal wound and excised. The proximal bowel was so packed with a thick, clay-like, fecal mass that it seemed wise to empty it before putting in a Paul tube. The peritoneum was closed around the two projecting ends of the bowel and the skin and muscles approximated by a few sutures of silkworm gut.

As the operation was not a lengthy one, very little shock followed. It was necessary, however, to remove the Paul tube on the second day as it became blocked with fecal matter. The wound became infected and the stitches were removed. The lad, however, was entirely relieved of pain, was able to take plenty of food, but did not seem to thrive. He became thinner and thinner. He was then taken to the roof of the hospital and kept in the open air. He improved immediately

and gained flesh rapidly. The wound healed up around the divided ends of the bowel and on April 15, two months after his first operation, a junction of the two free ends of the bowel was made. This was, unfortunately, not quite a success, as a small fecal fistula developed, which, however, gradually closed and he left the hospital on June 6.

He has reported at my office several times during the last year and his weight has increased more than 50 per cent. His present health is excellent.

Pathological Report. This was made by Dr. W. J. Deadman, pathologist of the General Hospital, Hamilton, and is as follows:

Gross appearance: The gross specimen consists of about seven inches of large bowel, in the center of which is a cauliflower-like growth completely encircling the bowel and causing partial obstruction of its lumen. This tumor mass is raised about three-fourths of an inch above the general level of the mucosa, and is papillomatous in appearance. The bowel wall beneath is apparently infiltrated by the growth, although the peritoneal surface has not been broken through. In some parts of the tumor there seems to be production of mucoid material.

Microscopic Examination. Histological examination of the tissue shows varied forms of atypical growth of gland epithelium. In some places there is an irregular formation of gland acini, many of which are lined by high columnar epithelium, while others show a marked heaping up of the epithelial cells. Some of these acini are distended and cystic, while in other places there is invasion of the underlying tissue by solid strands of epithelial cells. The interstitial tissue shows some areas of infiltration by polymorphonuclear leukocytes. In one area several epithelial cells are seen in mitosis.

Diagnosis. Adenocarcinoma.

In patients where there is an old chronic obstruction the fecal matter above the obstruction becomes so thick and difficult to get rid of that it will not go through a Paul tube. In a number of such cases I have made use of a suggestion of the late Dr. McGraw, of Detroit. He mobilized the growth, brought the

mass outside of the abdominal wound and placed his elastic ligature through the two healthy portions of the bowel above and below the growth. Instead of using his ligature, and where it is possible to milk back the contents of the proximal bowel I make a similar anastomosis with sutures, remove the mass, close the divided end of the lower segment and leave the proximal end open for drainage. This acts as a safety valve and gradually contracts and is easily closed later.

PERNICIOUS ANEMIA WITH SPECIAL REFERENCE TO THE SPLEEN AND THE LARGE INTESTINE

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Pernicious anemia is characterized by progressive degeneration of the red blood cells, permanent change in the method of the production of blood, marked changes in the spinal cord, achlorhydria and glossitis. The relation of the cord changes, achlorhydria and glossitis to the anemia has not been determined. The view held formerly, that the conditions were dependent on the anemia, is scarcely borne out by the fact that the symptoms are found in the earlier stages of the disease, neurologic examination showing that the cord changes may be as characteristic at any stage of the disease as the blood picture. The etiology of pernicious anemia is unknown, the early symptoms are indefinite, and by the time a diagnosis can be established the condition is incurable.

It has been asserted that certain cells found in the blood of patients with pernicious anemia are characteristic, but most observers agree that the pathologic change is in the blood as a whole, rather than in the development of specific types of blood-cells. While it is known that the actual perversion of the red-cell formation lies in the bone marrow, few believe that the cause originates therein. One might say that the bone marrow of a patient who has died from pernicious anemia furnishes only presumptive evidence of the disease. The pathologist would indeed be enthusiastic who asserted that in anemia other than of the aplastic type he could make a diagnosis of pernicious anemia

from the bone marrow examination alone. The diagnosis of pernicious anemia is by no means certain in the early stages. Most diagnosticians have had under their care patients believed to have pernicious anemia in the early stages who recovered with or without treatment. All have seen patients in whom the disease was so insidious in its manifestations that it had reached the incurable stage before its existence was suspected. Giffin, Woltman and Szlapka now have under observation a number of cases in which there are achlorhydria and some evidences of changes in the cord and tongue, but as yet without anemia. It will be interesting to learn in what percentage, if any, of these cases pernicious anemia develops.

The Relation of the Spleen to Pernicious Anemia. The relation of the spleen to the formation of the blood led the earliest observers of pernicious anemia to suspect that the spleen was associated with the disease. It remained for Eppinger to suggest that the removal of the spleen might cure, and early reports give testimony of temporary relief after splenectomy sufficient to justify a fair trial of the operation. Considering the confusion which so often attends the early diagnosis, it seems probable that obscure cases of hemolytic icterus and splenic anemia may have been accidentally included in the pernicious anemia group, especially since the pathologic changes in the spleen in pernicious anemia are not characteristic of the disease. Removal of the spleen in such cases has possibly given the impression that splenectomy may cure pernicious anemia. It has been a tradition of the medical profession that recovery of a patient in the face of a diagnosis of pernicious anemia proves the diagnosis wrong, and this conviction is still so strong as to prevent our readily accepting reports of cures. Even a superficial examination of the literature on pernicious anemia brings out in striking manner the fact that any form of treatment for pernicious anemia may be beneficial, or at least may appear to be beneficial. Patients with pernicious anemia have their ups and downs, and in an occasional case in which the diagnosis seemed to have been established by physicians of repute, there has been recovery which has lasted for years. Eppinger's work at least demonstrated that the removal of the

spleen initiated striking temporary improvement. In the cases observed in the Clinic there was marked gain in weight, and improvement in hemoglobin on an average of from 36 to 72 per cent., and the red-cell count increased on the average from less than 2,000,000 to more than 4,000,000.

Previous to November 1, 1917, fifty splenectomies had been performed in the Clinic for definitely established pernicious anemia. The operation was then discontinued almost entirely for three and a half years. Since there was not sufficient evidence that splenectomy would cure these patients, and since at that time the procedure was not known to give a degree of palliation sufficiently greater than that following blood transfusion, it seemed wise to await the verdict of time. Giffin and Szlapka have recently investigated the condition of these fifty splenectomized patients, and the pessimism which existed in the Clinic has been partly dispelled, at least. It was found that 21.3 per cent of the patients with pernicious anemia survived the operation three years or more, living two and a half times as long as the average in a similar group of nonsplenectomized patients at the same stage of the disease, and that 10.6 per cent are alive after more than five years. This clearly indicates in at least one-third of the cases that the average life of patients with pernicious anemia is greatly prolonged, and in about 10 per cent the prolongation is sufficient to lead to the hope that cures may result in some cases. We must take into consideration, too, the fact that no allowance was made for the normal mortality for the age period; all the patients who died were considered to have died from pernicious anemia. In the literature I have not found a comparable group of nonsplenectomized pernicious anemia patients who have done as well. The results in these fifty patients have been much better than we expected they would be when we abandoned the operation; they suggest that in certain early cases, of a type as yet not fully recognized, splenectomy offers a possibility of cure. Not only is this true, but also in the average cases the palliation following splenectomy is much greater than we have been able to obtain by blood transfusion. Transfusion gives temporary benefit. It seems to furnish red cells which may live and function for one or

two months or more, but there is not as yet evidence that the actual prolongation of life by that means is considerable. The blood picture improves after splenectomy, but the achlorhydria continues, and careful examination of the nervous system does not show changes for the better in the physical condition of the nerve structure, although the patients are much better in this respect clinically. A perhaps comparable showing was made by Stokes, in the Clinic, who found that careful treatment in syphilis of the nervous system made 86 per cent of the patients self-supporting, although the neurologists on examination were not able to find that there had been any marked change in the physical condition of the nervous system. Perhaps a lesion in the spinal cord gives physically the same signs whether the process is active or whether it is the scar of a former lesion. Clinically an ulcerating syphilitic lesion of the skin is more troublesome than the scar of a healed lesion.

The course of pernicious anemia following splenectomy, generally speaking, is ameliorated, the relapses are less severe and the cord changes less rapid than after transfusion or any other form of treatment with which we are acquainted. One of the functions of the spleen is to destroy deteriorated red cells. Apparently in pernicious anemia the red cells are born feeble and the spleen sacrifices red cells which, although imperfect, are the best the patient can produce and are capable of function. Removal of the spleen stops this destruction, but does not greatly affect the disease otherwise. The cases which seem to be most favorable are those in which hemolysis is most active, and those least characteristic of the disease. It also seems certain that early cases give a better prospect of benefit than late cases. When the disease has advanced to the point at which the bone marrow has been injured beyond the power of recuperation, the most to be expected is a temporary abatement of the symptoms.

Pathologic examination of the spleens we have removed has been rather disappointing. If we might assert that in the cases of pernicious anemia in which splenectomy was performed characteristic pathologic changes are found in the spleen, or that greater clinical improvement followed splenectomy in pernicious

anemia when the spleen was greatly enlarged, we could establish a definite relationship between the enlarged spleen and the disease, but the size of the spleen did not seem to bear any definite relationship to the severity of the disease; necropsies after death from pernicious anemia have often shown the spleen to be small, but during the life of the patient the spleen is usually enlarged. The average weight of the spleens removed in our cases was 400 gm. (195 gm. normal, Sappey) exclusive of two large spleens, one of which weighed 1600 gm. and the other 2220 gm. It seems probable, therefore, that during the course of pernicious anemia the spleen is enlarged, but that this enlargement is reduced to a certain extent in the terminal stages of the disease. The spleens removed during the earlier stages of the disease, therefore, would show an average greater weight and size than would be found at necropsy. In only two of our cases (both terminal) was the spleen below normal size. In pernicious anemia of the aplastic type the changes in the bone marrow are most marked and the spleen small. Whether this has any significance I do not know.

The question is not yet answered whether pernicious anemia is a definite entity or whether it is a terminal change of several conditions, and recognizable as pernicious anemia only when the patient has reached the state which we know will eventuate in death. If we assume that the removal of the spleen has a beneficial effect, as appears from the evidence at hand, are we to assume also that the spleen is the cause of the changes in the bone marrow? A study of the literature and of our cases does not give this impression. It would be more reasonable to assume that the same agents which destroy the bone marrow injure the spinal cord, produce the achlorhydria and the glossitis, affect the spleen, and that by removal of the spleen a vicious circle is interrupted.

The operation of splenectomy for pernicious anemia is simple. Among the first nineteen patients we had three deaths. The patients were operated on during crises as a last resort in very advanced cases. The best plan is to transfuse once or twice in order to get the patient on the up-grade before operating and not to operate during a crisis. We have had no operative death in the last forty cases.

The Possible Relation of the Large Intestine to Pernicious Anemia. Many observers have expressed the belief that toxic materials from the gastro-intestinal canal are the cause of pernicious anemia, and if this is true the probable location of this absorption would be in the proximal half of the large intestine. In a former communication[1] I discussed the physiology and pathology of the right half of the large intestine, my interest being excited primarily by the frequency of surgical tuberculosis and carcinoma of the large intestine, and still further by the work of Lane on intestinal stasis, which, like Banquo's ghost, will not down. There is a mystery concerning this portion of the human anatomy which conceals some queer metabolic disturbances, possibly, as I pointed out before, the result of throwing protein end products, which quickly undergo putrefactive changes, into an organ like the proximal absorbing half of the colon which primarily has an herbivorous function. Carcinoma or tuberculosis of the proximal half of the large intestine produces an anemia, unexplained by hemorrhage, much more severe than the anemia of carcinoma or tuberculosis of any other part of the body. Carcinoma of the fundus of the stomach perhaps is the only condition which produces an unexplained anemia which is in any way comparable. Tumors in the right half of the colon which are curable by operation may cause a reduction of the hemoglobin to below 25 per cent., edema of the lower extremities and a general physical condition which would apparently place the patient beyond surgical interference. Experience with a large number of resections for the relief of such patients in wretched general condition has shown a comparatively low mortality and a high percentage of permanent cures.

The flaw in Lane's assumption that the colon is the source of toxins is the failure to find evidence of disease on removal of the colon, beyond adhesions, dilatation and atony. Yet the same is true of exophthalmic goiter; the thyroid tissue of the hyperthyroidism is, strictly speaking, not so much abnormal as hypertrophied and superactive.

[1] Mayo, W. J.: Resection of the first portion of the large intestine and the resulting effect on its function, Jour. Am. Med. Assn., 1914, lxiii, 446–449.

Some very interesting observations on Balantidium coli as the cause of blood and cord changes of the pernicious anemia type have been made by Logan, of the Clinic, and, in a way, the cases studied support the hypothesis of the intestinal origin of pernicious anemia. The Balantidium coli is a flagellate parasite which rarely inhabits the intestinal tract of man, but is common in the pig. In man it is found in the intestinal wall, the bloodvessels, and other organs; it has the power of encysting and produces ulcers. The symptoms and findings of pernicious anemia, or of an anemia which cannot be distinguished from it, accompany the chronic type of the infection. Eosinophilia is not present in the chronic cases. Glaesseur has found a hemolytic agent in Balantidium coli. Since this parasite penetrates the intestinal wall and its bloodvessels and other organs of the body, and encysts, it is impossible to be sure that the organism is not harbored in the tissues of the body simply because it has been eradicated so that it is no longer found in the stools. Thus, if the pernicious anemia syndrome persists, it cannot be determined whether the Balantidium coli hemolysin is still active, or whether once the complex is started it becomes an entity. In any event the pernicious anemia of Balantidium coli progresses to a fatal issue. The number of observed cases is small and the pernicious anemia might have been a coincidence. Honest card players have been known to hold four aces.

I fully realize that there is little evidence to show that the etiologic agents of pernicious anemia arise in the large intestine. Pernicious anemia annually destroys more than 6000 lives in the United States. Even though the disease is common, we have learned comparatively little concerning it during the past generation. It behooves us to keep an open mind, to study carefully the blood conditions of all patients who have disease of the large intestine, to try to ascertain whether anemia, achlorhydria, glossitis or some other abnormal condition exists, and especially to study carefully all cases in relation to cause and effect in which the large intestine is removed or in which splenectomy is performed.

THE ALTERED ANATOMY AND PHYSIOLOGY OF THE CECUM AND ASCENDING COLON, THE RESULT OF ADHESIONS

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The importance of adhesions involving the abdominal viscera in the causation of symptoms of various degrees of severity has long been recognized. In the past, however, these symptoms have often been discussed in a general way, and the results of abdominal adhesions have been often considered as consisting principally of indefinitely localized abdominal pain and more or less disturbance of the functions of the gastrointestinal tract. An exception to this viewpoint has, of course, always existed in those cases in which there has been acute obstruction of some portion of the intestinal canal.

Gradually, however, the fact has become established that the term "adhesions" is not sufficiently clear; that we must try to separate as clinical entities those groups of cases in which the adhesions affect principally the anatomy and physiology of a definite portion of the intestinal tract.

Moreover, I consider it to be established that adhesions involving the cecum and ascending colon to such an extent as to cause symptoms can often, indeed, as a rule, be correctly diagnosed; that they give a distinct symptomatology, and that a definite line of treatment, surgical or otherwise, is indicated when such a diagnosis is made.

Any consideration of the treatment or symptoms of such derangements must rest upon a consideration of the variations

from the normal anatomy and physiology of the parts affected, and upon certain factors concerning the nature and origin of the adhesions we have to deal with.

Normally the ascending colon is the largest portion of the intestine, the cecum being its largest portion. In the majority of cases the cecum has a freer range of motion than the other portions of the large gut, the ascending colon being, as a rule, fixed in the right iliac fossa and right kidney space. In but 25 per cent. of cases is there a distinct mesocolon for this portion of the intestine.

Physiologically, two points are to be noted as regards the ascending colon: (1) Its contents are semifluid or fluid and it is here that a large part of the water from the fecal stream is absorbed. (2) It is characterized by antiperistalsis, the wave being stopped by the closed ileocecal valve. This reversed peristalsis has for its object the thorough mixing of the food contents before the withdrawal of the fluids. When the mixture is right, true peristalsis replaces the antiperistalsis, the food is pushed along to the transverse colon, and the ileocecal valve opens for the admission of a fresh supply of material from the small bowel. It must be realized that in this lift of material alone, the individual being in the vertical position, considerable work is done by the musculature of the cecum and ascending colon, and even a slight additional burden caused by pathological conditions may render normal function impossible.

The ascending colon, by its location, structure and function, is not much affected by acute inflammatory conditions arising within or from it. The mechanical effects of occlusion by neoplasm are well known and need not be discussed here. The most frequent cause of malfunctioning of this portion of the gut is adhesions. Excepting those cases in which we find adhesions within the abdomen following upon a definite infectious peritonitis—in which event the adhesions naturally correspond in location to the site of this process—the right iliac fossa is by far the most frequent site of those bands, folds and adhesions with which we are now dealing.

In a previous communication[1] I have laid stress upon the fact that these bands, folds and adhesions are, as a rule, not the result of faulty development or of failure in the descent or rotation of the gut, but are inflammatory, caused by a peritonitis, acute or chronic, recognized or unrecognized, in adult life, infancy or the prenatal period. I believe that a recognition of this fact will do much to do away with the hesitation with which many surgeons approach the cure of conditions resulting from them, and with faulty surgical procedures which attempt an approach at correction from the wrong angle. The surgeon who deals with such adhesions must attempt to relieve symptoms due to them and to prevent their recurrence, not to reconstruct an intestinal canal according to his ideas of its physiology. Since these adhesions are, as a rule, inflammatory, they produce alterations of greater or less degree in a gut previously normal or nearly so. The inflammatory process causing them may have occurred at any period, and may not, indeed frequently does not, have symptoms severe enough to justify a clinical diagnosis of peritonitis. Its results are, however, of the same nature, if not the same degree, as those caused by a definite purulent peritonitis; *i. e.*, abdominal adhesions as distinguished from those possibly non-infectious agglutinations, transient in nature, referred to by certain authors.[2] In other words, the results involve destruction of the endothelial layer of the serosa, with subsequent formation of permanent pathological structures binding together certain of the abdominal viscera.

To approach correctly the treatment of such adhesions involving the cecum (with the ileocecal junction and the ascending colon), we must consider their grouping: (1) As to their extent and their location; (2) as to their anatomical effect by traction upon the abdominal viscera; (3) as to the systemic and local symptoms produced.

1. Since the degree of frequency and extent of the peritonitic process causing these adhesions varies so greatly, it follows that

[1] Ross and Mencke: Am. Jour. Med. Sc., February, 1917, No. 2, cliii, 261.
[2] Hertzler, W.: Tr. Western Surg. Assn., Minnesota, 1916, xxvi, 99–141.

in a given case the adhesions may be of the slightest extent or so dense and extensive as to almost obscure the normal anatomy of the parts. Strange as it may appear, a few small adhesions may by their location cause a far more marked symptomatology than far more evident structures not so disadvantageously placed. Adhesions seriously interfering with the ileocecal junction, causing fixation when the rest of the intestines in this region are ptosed, give probably greater distress than far greater structures obstructing only the cecal lumen.

2. Adhesions about the cecum and ascending colon do not necessarily give symptoms confined to these structures. They may cause interference with the gastrointestinal tract in any of its portions. We must consider among other conditions: (*a*) Malposition of the stomach associated with ptosis and fixation of the transverse colon, caused by adhesions; (*b*) traction upon the duodenojejunal junction due to adhesions in the ileocecal area;[1] (*c*) possible gastric lesions of an organic character superinduced by malposition caused by adhesions lower down;[2] (*d*) traction upon the splenic and hepatic flexures by the misplaced transverse colon and fixed ileocecal region.

3. Bearing in mind such conditions as these, we may approach a rational attempt at classification of the symptoms due to interference with the cecum and ascending colon.

The local symptoms are: Pain about the ileocecal junction and hepatic flexure, more or less constant with occasional marked exacerbations; tenderness, not localized to McBurney's point, but more diffuse; a feeling of actual stoppage of intestinal contents, *especially gas*, at the hepatic flexure. Fluoroscopic examination often displays a fixed point at the ileocecal junction, hepatic flexure or both, with delayed passage of intestinal contents. There is frequently a constriction, posterior displacement and fixation of the terminal portion of the ascending colon and hepatic flexure. Often there is an associated dilatation of the cecum and first portion of the ascending colon.

[1] Lane, A. W.: Lancet, London, 1919, cxcvi, 1, 333.
[2] Ibid.

As referred local symptoms, must be mentioned especially the gastric ones; *i. e.*, so-called dyspepsia without evidence of gastric disease, *but with tenderness in the right iliac fossa* often manifested, vomiting without gastric or upper abdominal cause and associated with fluoroscopic evidence of disturbed physiology of the cecum and colon.

The functional symptoms are due to delay in the handling of the intestinal contents by the large gut—constipation, intestinal torpor and distress due to the excessive formation of gas and its slow and painful progress along the gut tube. The systemic symptoms are directly due to this coprostasis, with the excessive absorption of putrefactive materials. They may range from mere weakness, malaise and listlessness, cold feet and hands, and chronic headache, to the most extreme depression with secondary joint and other lesions due to what is practically a focal infection. Fortunately these severe cases are rare, for they are most intractable under any form of treatment.

It seems to me that careful study of a given case, and a consideration of the local and general symptoms, with careful *x*-ray studies, will in most instances enable us to make a correct diagnosis of cecal (ileocecal) and colonic adhesions.

Our ideas upon treatment should take just as definite a form.

It can be broadly stated that the cases, insofar as treatment is concerned, fall easily into three groups:

1. Those cases in which the symptoms, due to a minor grade of adhesions, accompany a general ptosis of the viscera in individuals of the so-called "neurasthenic type." In these instances the difficulty caused by the adhesions is not the patient's major ailment. Attempts at surgical cure of such patients are rarely successful and have been in the past a cause of much unfavorable comment upon the surgery of intestinal conditions in general.

2. Those cases in which the adhesions themselves are the main or only cause of the symptomatology, giving rise to those local and general symptoms previously described. Either the local or general symptoms may predominate. If the local evidences are not marked, and the systemic effects are grave,

careful study is necessary not only to demonstrate the cause of the general symptoms but also to eliminate those cases in which the intestinal lesion but accompanies other underlying factors.

3. Those cases with most severe general symptoms, in which the visceral conditions are such as to call for extensive surgical procedures—such as resection of the colon. Fortunately such cases are extremely rare. The primary mortality is high, and while there have been occasional brilliant results they have been so few as to discourage frequent attempts at such surgery.

My conclusions as to symptoms and treatment of the lesions under consideration are based upon the observation of a considerable number of cases.

I have been able to collect from various hospital services the records of 36 cases I have operated upon since I have made routine examinations for abdominal adhesions involving the colon and ileocecal region whenever an abdominal operation is performed. The records are by no means uniform and it has been difficult to follow some of the cases. I have classified the 36 cases as follows:

1. Those in which the main pathological condition was an ileocecal interference due to a Jackson's membrane or Lane's kink, with or without chronic appendicitis. There were 9 such cases.

2. Cases more correctly to be classed only as "adhesions" involving the ascending colon and ileocecal region. These were 14 in number.

3. Cases in which a major surgical condition associated with those under consideration were also operated upon. These were 3: 1 case of cholelithiasis; 1 case of salpingo-oöphoritis; 1 right inguinal hernia.

4. Cases of abdominal adhesions (3 classed as Jackson's membrane) in which the records were insufficient to permit of proper analysis.

Of the 9 cases of Jackson's membrane in which adequate records were found, 8 had an appendectomy performed at the same time, 1 had had an appendectomy four years previously

with no benefit. In 4 of the 9 cases a diagnosis of adhesions involving the ascending colon or hepatic flexure was made before operation; in 2 of these Jackson's membrane was definitely noted as a cause.

Symptomatically the cases resembled each other in all having a history of abdominal pain. Usually this pain suggested perhaps appendicitis, subacute in type. An occasional patient (F. K. M.) would describe more in detail symptoms pointing to partial bowel obstruction and stasis rather than those leading to a diagnosis of inflammatory disease. One case had joint and toxic symptoms of long standing—these were entirely relieved by the operation.

In 8 of the 9 cases a record of the ultimate result is at hand: 6 were cured (1 still complaining of constipation); 1 (Mrs. Geo. I.) was benefited ("partially cured," September 30, 1920); 1 was benefited, but still seems to be in very poor shape. The operative recoveries in all of these 9 cases were uneventful.

In all of the cases the appendix when still present was removed, the adhesions were freed, the surfaces covered with peritoneum where possible and swabbed with paraffin (or olive) oil.

There were 14 cases of adhesions. Of these 9 were cured with possible minor symptoms remaining; 2 were improved; 1 was unimproved; 2 died.

An analysis of these successful cases demonstrates that attention to the local pathology (appendicitis, etc.), with release of adhesions only and measures to prevent their recurrence, often results in a cure. It has not been found necessary to perform resections, plications or intestinal anastomoses. There can be no doubt that more extensive operations would have been associated with greater mortality—the results could hardly have been more satisfactory.

In the one totally unsuccessful case a dense band of adhesions involved the ascending colon, causing angulation and fixation. It is also noted that the stomach was ptosed to below the umbilicus. The patient developed a slight hernia through the upper angle of the incision. Nine months after the first operation she

was operated upon by another surgeon – with what result I do not know.

Among the cases much benefited (*i. e.*, cured) the following brief note of the pathology, symptoms and treatment may be given:

Pathology.	Symptoms.	Treatment.
1. Sigmoid tied to lateral abdominal wall; chronic appendicitis	Upper abdominal	Appendectomy; release of adhesions.
2. Adhesions of omentum and almost complete obstruction of ascending colon; chronic appendicitis	. . . . .	Appendectomy; release of adhesions.
3. Subacute appendicitis; subacute peritonitis; young adhesions RIF	Lower abdominal, plus nausea and vomiting	Appendectomy; release of adhesions.
4. Abdominal adhesions anchoring ascending colon and hepatic flexure; chronic appendicitis	Constipation	Appendectomy; release of adhesions.
5. Adhesions about head of cecum; chronic appendicitis	Pain, right lower abdominal segment	Appendectomy; release of adhesions.
6. Adhesions (location); chronic appendicitis	Apparent attacks of subacute appendicitis	Appendectomy; release of adhesions.
7. Retrocecal appendix; subacute; omentum constricting ascending colon, five years old	Attacks of appendicitis	Appendectomy; release of adhesions.
8. Previously operated; adhesion involving hepatic flexure and transverse mesocolon; anchoring stomach to right of midline	Generalized abdominal pain and pain on walking; constipation	Release of adhesions.
9. Previous operation; adhesions, ascending colon, duodenum and pylorus	Very general abdominal symptoms	Release of adhesions; resection of R. broad ligament.

Two cases of this series died: One with extensive colonic adhesions died of acidosis five days after operation. The second case died within forty-eight hours after operation. This patient was not in good condition before operation and had very extensive adhesions. She died with symptoms suggestive of peritonitis, confirmed at postmortem examination.

In connection with those instances in which the adhesions are extensive and the gut has been kept from functioning normally for a long period of time, it may be noted that for a number of

days immediately after operation the patient may suffer from symptoms suggestive of toxic absorption. The relaxed and poorly muscled intestine cannot propel the fecal material quickly enough and temporary stasis with excessive absorption of harmful materials takes place. When this becomes extreme an acidosis, perhaps fatal, supervenes. Measures to combat this possible condition should be undertaken immediately after operation in every instance. Sir A. Lane attributes some of these symptoms to interference with lymphatic channels formed within the adhesions and advises drainage after extensive operations even when the intestine is not entered.

THE MANAGEMENT OF PELVIC ABSCESS IN ACUTE APPENDICITIS

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The lesions of an infective peritonitis of appendical origin are quite analogous to those of the soft parts. This is especially the case in the early stages of infection and it is only after the discharge or removal of the resulting abscess contents that a material difference may be noted. In the soft parts gravity unquestionably dictates the drainage of an abscess cavity at its most dependent portion. On the other hand, satisfactory drainage of abscesses, even of those involving Douglas' cul-de-sac, can be obtained through an opening in the anterior abdominal wall. This apparent contradiction, in the abdomen, of the law of drainage may be accounted for by the fact that the rhythmical contraction of both the thoracic and pelvic diaphragms constantly directs fluid abdominal contents toward any adventitious opening in the abdominal wall as long as the path of communication remains unobstructed. Furthermore, this same movement in the direction of least resistance is favored by the counterpressure resulting from the more or less intermittent involuntary contraction of the abdominal muscles themselves, as well as by the peristaltic activity of the smooth muscle fibers of the intestine. The resultant activity

of these different forces comprising intra-abdominal pressure accounts for the difficulty frequently experienced in preventing the prolapse of mobile abdominal contents through an operative incision and readily explains the drainage of inflammatory or hemorrhagic abdominal exudate through an opening in the abdominal wall.

This general "law," however, does not apply to all parts of the abdomen alike, for, in the upper part, drainage of the subphrenic spaces through an anterior opening is rarely satisfactory without siphon attachment. In this location the beneficial effect of the peristaltic activity of the small intestine is largely lacking, while the mobility of the adjacent diaphragm is seriously impeded if not entirely suspended by fibrinous exudate, and more especially by the pressure of the accumulated fluid.

Then, too, the overhanging osseous and cartilaginous thoracic wall, together with the adjacent solid and more or less fixed abdominal viscera, prevent the normal contraction and obliteration of the abscess cavity, which is ordinarily so deeply placed that its contents are more likely to be compressed upward against the diaphragm by the contraction of the abdominal muscles than toward any opening anteriorly.

Although quite analogous in their resistance to infections, the fact that the reparative power of the peritoneum is superior to the reparative power of the tissues of the abdominal wall cannot be too strongly emphasized. This is illustrated by the fact that while the discharge from the intraperitoneal portion of an abscess rapidly decreases in quantity and quickly assumes a healthy type, the divided tissues of the abdominal wall regularly undergo appreciable and occasionally extensive necrosis, associated with an abundant foul discharge that continues until healthy granulations are established. This contrast in the relative reparative power of the peritoneum and abdominal wall is perhaps most striking where, after the removal of an acutely distended but not perforated gangrenous appendix, the abdominal incision is closed without drainage. Although, in many of these cases, infection occurs in the suture line, the underlying

peritoneum ordinarily escapes. The reparative power of the peritoneum is also well illustrated in one of the cases of this series in which, after the evacuation of an appendix abscess of some days' duration, a loop of small intestine forming the wall of the abscess cavity was found to have so extensively sloughed as to require resection with an end-to-end anastomosis. Prompt healing occurred without the slightest indication of an intestinal fistula, the adjacent pelvic abscess being drained in the usual way.

Drainage of pelvic abscesses by the suprapubic route exclusively does not meet with universal approval. One year ago MacLaren, in a paper read before the American Surgical Association, advocated supplementing abdominal drainage with a counteropening through the rectum or vagina, such an opening, at times, immediately preceding the abdominal incision. In this paper he referred to the recently published report, by Rullison, of all cases operated on at the Presbyterian Hospital, New York, in 1916, in which no instance of this method of drainage was employed, with the comment that by its use in appropriate cases the mortality of 9.1 per cent might have been materially reduced.

Statistics such as Rullison's are always interesting, although, because of the inevitable differences in the operative and postoperative methods employed by different surgeons in the care of these patients, conclusions may prove very misleading. Such statistics, however, serve a useful purpose in providing an opportunity to judge the relative value of different methods of treatment and they should always be collated and arranged with this end in view. On the other hand, deductions based on a series of cases such as MacLaren's, in all of which the principles of treatment were identical, are not only interesting and valuable but highly instructive.

The fact that the writer has never utilized a counteropening through either rectum or vagina in the drainage of a pelvic abscess complicating acute appendicitis is in such marked contrast to the experience of MacLaren that it seemed to justify

the further consideration of this subject. With this object in view the writer has made a careful study and analysis of the treatment and results of 351 cases of this lesion that have come under his personal observation during the past twenty-one years (of which 314 are taken from the records of the Presbyterian Hospital). From this series are excluded a number of fatalities in desperate cases which scarcely survived the operation twenty-four hours and in which no form of drainage could have exercised any favorable effect. In the remaining 351 cases the gross pathology has varied according to the duration and type of the infection as well as the location of the appendix.

Prior to perforation an abundant, odorless, occasionally turbid, fluid exudate has been found, free from pathogenic organisms and reparative in character. In this group of cases primary union was frequently obtained either without drainage or where precautionary drainage for thirty-six hours only was employed. Occasionally infection of the suture line developed and rarely the escape of a purulent odorous collection on the withdrawal of the drain at the end of thirty-six hours necessitated its replacement. After perforation has taken place the exudate changes into foul, fetid pus, and, irrespective of the location of the appendix, drainage in these cases is always required. If the appendix overhangs the pelvic brim, pelvic abscess is inevitable. If the appendix is retrocecal, pelvic abscess is not infrequent, although there may be no apparent connection between it and the abscess about the appendix. It is quite evident, therefore, that in all cases in which the peritoneal cavity is opened for the removal of an acutely inflamed appendix the pelvis should invariably be searched for pus. Only in those cases in which a perfectly walled-off abscess may be opened without entering the peritoneal cavity should this search be omitted. In this latter group (no cases of which are included in this paper) the efficacy of anterior drainage is generally acknowledged.

The incision adopted in the treatment of this series of cases has been invariably the intermuscular. This has served both for the removal of the appendix and the drainage of the pelvic

abscess. The splitting of the muscle in preference to its division conserves the muscle strength of the abdominal wall, favors an early return of its power to contract and materially contributes to the restoration of normal intra-abdominal pressure. These forces together tend to check a spreading intraperitoneal infection and serve to conduct the flow of the purulent discharge to the drainage opening in the abdominal wall. By the use of this incision the wound heals more quickly and firmly with less chance of the development subsequently of a ventral hernia. Should such a hernia develop, its repair is both simple and satisfactory.

Attention to other operative details materially promotes the efficiency of subsequent drainage.

The removal of the appendix, and consequently of the source of infection, the insertion of a purse-string suture or, when that is impossible, the reinforcement of the appendix stump by the superposition of an omental flap, the minimum handling of the small intestine, the thorough evacuation of the pelvic abscess and the careful cleansing of the cul-de-sac with saline irrigation are important steps of the operation. The pelvis is irrigated with the shoulders of the patient elevated, the intestinal loop being held gently aside by the pressure of a long, narrow retractor against a previously inserted pad. When concluded, and the pelvis has been sponged dry, the drain is inserted to the bottom of the cul-de-sac before the withdrawal of the retractor and pad. After firm compression of the adjacent abdominal wall and bimanual compression of the right ileocostal space have either excluded or removed all further exudate, the abdominal incision is closed, rather loosely, around the projecting tube with interrupted chromic-gut sutures. The primary abscess cavity, if paracecal or retrocecal, is similarly drained through the same opening, or, if situated above the crest of the ilium, through a counteropening in the flank.

For about two years, in the first part of this period, the primary drain consisted of a Mikulicz tampon of plain or iodoform gauze, the incision being only partially closed. The severe pain occasioned by the withdrawal of this drain as well as by its replace-

Date.	Type of drainage.	Replacement drainage.	Number of cases.	Irrigation through primary drain.	Average number of days, cured.			Complications.								Cured.		Died.		Secondary abscess, case number.
					Uncomplicated.	Complicated.	Complications only.	Fecal fistula.		Phlebitis.		Respiratory.		Secondary abscess.						
								Actual.	Per cent	Actual.	Per cent	Actual.	Per cent	Actual.	Per cent	Actual.	Per cent	Actual.	Per cent	
Jan., 1900-Nov., 1900	(*a*) Mikulicz; (*b*) sterile gauze; iodoform gauze	Gauze packing	39	0	32	38	55	4	10	2	5	1	2.5	0	0	32	82	7	18	
Nov., 1900-Feb., 1904	(*a*) Iodoform gauze or sterile gauze and rubber tube; (*b*) Mikulicz rubber tube	Same as original	39	±	28	32	44	4	10	1	2.5	0	0	2	5	32	82	7	18	8138
Apr., 1901-Mar., 1905	(*a*) Rubber tubes; (*b*) rubber tubes and gauze wick	Cigarette or rubber tube	24	+	27	31	44	7	29	0	0	0	0	0	0	22	92	2	8	8179
May, 1902-Nov., 1908	Cigarette only	Same as original	33	0	24	24	30	1	3	0	0	0	0	0	0	32	97	1	3	
Feb., 1904-Dec., 1904	Rubber tube and cigarette	Same as original or rubber tissue	7	+	26	27	32	1	14	0	0	0	0	0	0	7	100	0	0	
Jan., 1905-1907	"Collar" cigarette	Cigarette	27	0	25	26	37	2	7.5	0	0	0	0	1	4.7	24	89	3	11	5135
Mar. and Apr., 1912	Rubber tube and cigarette	Cigarette or catheter	3	+	21	21	21	0	0	0	0	0	0	0	0	3	100	0	0	
1908–1912	Cigarette only	Cigarette or rubber tissue or catheter	72	0	20	23	44	4	5.6	1	1.4	1	1.4	5	7	71	98.5	1	1.4	7521 7507 7967 7758
June, 1911-present	Rubber tube and gauze wick	Catheter	70	+	20	23	41	4	5.7	1	1.4	5	7	2	2.9	68	97	2	2.9	9529 19472 M-43
Total.			314	..	..	..	..	27	8.6	5	1.6	7	2.27	10	3.2	291	93	23	7	

ment, together with the risk of a prolapse from the rupture of intraperitoneal adhesions (which actually occurred in one instance at the initial dressing five days after operation), led to its being discontinued and to the substitution of a rubber tube enmeshed in gauze. It is quite possible that the use of an investing rubber dam would have eliminated both the physical discomfort and the risks of the Mikulicz tampon. It has never been employed, however, as other methods of drainage have proved thoroughly satisfactory.

In 1902 two or more cigarette drains were substituted for the enmeshed rubber tube, and this method of both primary and secondary drainage was continued for ten years. During this same period the combined use of a cigarette drain and rubber tube was occasionally employed, and for three years a cigarette drain inserted through a rubber tube equal in length to the thickness of the abdominal wall was quite generally used. This rubber "collar" was designed to facilitate drainage by preventing too sudden approximation of the separated muscle planes.

In 1911 laboratory investigation demonstrated that capillary drainage by means of a cigarette drain was practical for the serous but not for the cellular part of purulent material, and that within a few hours its drainage value became greatly impaired even for serous discharge. This method was therefore discarded, although it had generally given satisfaction, in favor of the simple rubber tube containing a narrow wick of gauze projecting slightly at either extremity, in order that whatever advantage might accrue from the use of capillary drainage in the first few hours after operation might be retained. The withdrawal of this form of primary drain as well as that of the tampon and cigarette drains, at the end of four or five days, invariably releases an appreciable amount of pent-up purulent material which with ideal drainage should have previously escaped into the dressing. It has further been learned that this undesirable blocking of the discharge can be avoided only by the removal of the drain within the first thirty-six to forty-eight hours after the operation. That such a simple remedy was not immediately adopted was due entirely to the difficulty experienced in the

insertion of the replacement drain before the end of the fourth or fifth day—a difficulty that was eventually overcome by the use of a Nélaton catheter. The rounded extremity and the flexibility of this instrument facilitate an easy, painless passage along a recently formed sinus leading into an abscess cavity, while the fact that the gauze in which its extremity is invested is stained at some distance from the edge of the incision with discharge conclusively demonstrates its value as a drain. At each dressing the character of the discharge from the deepest part of the abscess cavity can be determined by compression of the extremity of the catheter just prior to its withdrawal, and by the use of similar siphon action the entire abscess tract can be rendered practically dry after irrigation. The only reason why the catheter is not used as a primary drain is that, on account of its very flexibility, the passageway leading into the cul-de-sac would probably be much more sinuous (and therefore less suitable for the effective discharge of the abscess contents) than the much straighter channel secured by the use of a relatively unyielding rubber tube for the first thirty-six or forty-eight hours. The catheter, then, is used both as a drain and for purposes of irrigation, the latter begun four days after operation and continued until the offensive character of the discharge disappears. At the end of eight to ten days the intraperitoneal part of the sinus is practically obliterated and the closure of the superficial wound follows in a few days. To sum up, the establishment of a direct passage into the deepest part of the abscess cavity is obtained by the use of a medium-sized rubber tube for thirty-six hours, and the continued unobstructed patency of this passage is secured by the daily introduction of a Nélaton catheter. So satisfactory has this method of drainage proved that its use has been extended to the drainage of infectious processes in all parts of the abdomen with good results.

While the value of effective drainage in these cases must not be minimized, the ultimate closure of the abscess tract is unquestionably hastened by irrigation. As this practice has become more and more a matter of routine the average duration of healing has progressively decreased from thirty-two to twenty days.

SECONDARY ABSCESSES

Date.	Case No.	Type of drainage.	Replacement drainage.	Postoperative development (day).	Location.	Amount.	Character of pus.	Treatment.		Result.	Operative findings.	Autopsy findings.
								Incision.	Spontaneous rupture.			
Feb., 1902	8138 (A. W.)	Mikulicz and two rubber tubes into pelvis	Same	14–21	Pelvis	2 oz.	Different from abdominal pus	....	....	Died 23 d.	.....	Pelvic abscess about 2 oz.; general peritonitis purulent in character and of a different character from pelvic abscess
June, 1902	8179 (H. S.)	Mikulicz and rubber tube	Same	4	Pelvis	1 pint	Thin	At dressing a drawn up death nine hours later	loop of gut into wound; and a half	Died 5 d.	Finger in pelvis entered a cavity containing 1 pint of thin pus; no kinks or obstruction of intestines	
April, 1907	5135 (M. C.)	"Collar" cigarette	Same	7	Behind and to left of rectum	1 oz.	Foul	Exploratory; ileostomy and drainage	....	Cured 51 d.	Small intestines of dull color; fibrinous flakes; a distinct band in iliac fossa, constricting gut; abscess and foul pus	
1908	7521 (E. M.)	Cigarette	Same	5–7	Abdominal wall; right lumbar	1 oz.	Yellow	Right flank	....	Cured 29 d.		
1908	7507 (J. B.)	Cigarette	Same	12–17	Peritoneal left lower quadrant	2 oz.	Streptococcus	Left lower quadrant	....	Cured 44 d.	Mass not palpable per rectum.	
1909	7967 (J. W.)	Cigarette	Same	7	Left iliac fossa into pelvis	2 oz.	Streptococcus	Left rectus	....	Cured 27 d.		
1909	7758 (K. D.)	Cigarette	Same	12–19	Abdominal wall, left lower quadrant	+	Streptococcus and colon	Left lower quadrant	....	Cured 34 d.		
1909	9529 (W. G.)	Cigarette	Same	12–17	Pelvis	++	.....	....	Original incision	Cured 40 d.		
1914	19472 (F.)	Rubber tube and gauze wick	Catheter	20–38	Subphrenic	3 oz.	Streptococcus and colon	Original incision enlarged (McBurney)	....	Cured 60 d.-21 d.		
1914	(M–43)	Rubber tube and gauze wick	Catheter	8	Above and to outer side of cecum	+	.....	....	....	Cured.		

Irrigation also contributes to the comfort of the patient by mitigating the offensive character of the discharge.

The fatalities occurring shortly after operation in this series of cases must be ascribed to the intensity and extent of a far-advanced peritonitis which in no way could have possibly been diminished by any method of drainage. A study of the results obtained in all other cases seems to justify the conclusion that drainage through a rectal or vaginal counteropening would not have affected materially the mortality. To this statement exception should be made in two instances in which the Mikulicz tampon was employed: In one, twenty-three days after operation, an autopsy showed a collection of two ounces of pus in the pelvis, markedly different in its appearance from the generally distributed purulent exudate of associated diffuse septic peritonitis; in the other, a loop of small intestine prolapsed on the fourth day after operation at the time of the initial withdrawal of the tampon. A rapidly developing peritonitis then led to a secondary laparotomy with the discovery of at least a pint of thin pus in the pelvis. The patient did not long survive the operation.

In cases drained by cigarette drain, between 1902 and 1912, secondary abscesses developed in five cases. These appeared as early as the fifth and as late as the nineteenth day after operation. The location of the abscess in one instance (not included in table) was in the neighborhood of the gall-bladder; in another, in the right lumbar region; in two instances, in the left lower quadrant near the anterior parietal peritoneum, and in the remaining three, in the left side of the pelvis—in only one of which was the abscess palpable through the rectum.

Six of these secondary abscesses were opened through an anterior incision. In one, the abscess discharged spontaneously into the original abscess tract. All recovered, and in only one patient—in whom, in addition to an abscess in the left side of the pelvis, the small intestine had become constricted by a fibrinous band—was the condition serious. A prompt recovery, however, followed the evacuation of the secondary abscess and an ileostomy for the relief of the obstruction.

Since 1912, during which period primary drainage by a rubber tube containing a small wick of gauze has been employed, two instances of secondary abscess have developed: one above and to the outer side of the cecum, and the other in the right subphrenic space. In both, recovery followed the evacuation of the pus through an appropriate incision. There have been no instances of the formation of a secondary abscess in the pelvis.

Of all secondary abscesses complicating appendicitis those occurring in a part of the abdomen remote from the pelvis must be ascribed to failure to remove all infectious foci, and it is reasonable to infer that those appearing in the left lower quadrant near the anterior abdominal wall are due to a similar cause. Those occurring in the deeper part of the pelvis, especially where an elastic swelling can be palpated through the rectal wall, probably indicate defective drainage. In those that have no connection with the original drainage tract the cause may at least be said to be doubtful.

Further valuable evidence of the efficiency of pelvic drainage is derived from the results of two autopsies occurring respectively thirteen and eleven days after operation. In these cases a collar cigarette drain, and two cigarette drains, respectively, had been employed. In neither was any accumulation of pus found in the pelvis. The case reports also show four instances of secondary enterostomy done six, seven, six and six days respectively after the primary operation. In one, already referred to, there was a collection of pus on the left side of the pelvis near the rectum, having no communication with the original drainage tract. In the remaining three the pelvis was entirely free from purulent exudate. In three of these cases a collared cigarette drain and in the fourth a rubber tube containing a small wick of gauze had been used. The sole fatality occurred in one of the cases drained by a collared cigarette.

Analysis of the evidence presented by these different conditions justifies the conclusion that, with the exception of the two cases treated by Mikulicz tampon and two instances of secondary abscess in the deeper part of the pelvis, the drainage of the

CASES REQUIRING SECONDARY OPERATION—NOT FOR ABSCESS

Date.	Case No.	Primary drain.	Secondary drain.	Type of operation.	Operative findings.	Result.	Autopsy findings.
June, 1900	7735 (E. M.)	Iodoform gauze	Same on second day	Exploratory median incision on fourteenth day	Considerable serum in general cavity; no odor and no pus; intestines congested	Died on twenty-first day.	
Dec., 1901	8086 (M. B.)	Mikulicz and rubber tube; wrapped in iodoform gauze; no pelvic abscess	Same	Exploratory median incision on fourth day	Intestines somewhat congested; were separated till bottom of pelvis was exposed; no abscess; a Mikulicz tampon of iodoform gauze to pelvis	Died on fifth day.	
Mar., 1906	1960 (A. F. I.)	"Collar" cigarette	.....	(*a*) Intestinal obstruction on fifth day; (*b*) enterostomy second day	(*a*) Serous fluid in pelvis without odor; kink in small intestine relieved; no special relief. (*b*) At first great relief, through free evacuation, both through fistula and rectum	Died on eleventh day	Around stump of appendix no sign of either ligature; a small black sloughy fistula 4½ feet above valve; no gangrene in either large or small intestine.
Nov., 1907	5596 (A. B.)	Cigarette to pelvis; "collar" cigarette to iliac fossa	Fourth day, all out	Ileostomy on sixth day; first incision left ileocostal space; second incision midline above umbilicus	Small amount of free serous fluid; the walls of small intestines showed a number of fragments of plastic material; exploration of pelvis showed no accumulation of pus	Cured by thirty-third day.	
1909 . .	9772 (S. H.)	Cigarette	Gauze packing because of hemorrhage	Twelfth day, considerable hemorrhage after dressing	.....	Died on thirteenth day	Small pockets of pus between matted coils of small intestines; no collection of pelvic pus; tubes and ovaries normal.

cul-de-sac through an anterior opening has been satisfactory. It is at least not too much to state that during the past ten years, since the inauguration of the present method of drainage, the absence of a single instance of secondary formation of abscess in the pelvis seems to exclude the need of the use of a rectal or vaginal counteropening.

The possibility of inefficient drainage either of the cul-de-sac or of some contiguous part of the pelvis, however, must always be kept in mind. To forestall surprise, a daily leukocyte count should be taken and a rectal examination should be made at least once in forty-eight hours in search of tumefaction or tenderness. It is both interesting and important to note that usually both of these symptoms may be elicited. The tenderness, however, is decidedly less than prior to the operation, and the sense of swelling—the result probably of the matting together of intestine and omentum—gradually subsides and entirely disappears in the course of a few days.

By one without experience in the use of rectal or vaginal drainage, any criticism or discussion of its value is out of place. Perhaps the possibility of the entrance of additional causes of infection through such an opening may be advanced as a theoretical objection. Furthermore the possibility of a persistent sinus must not be overlooked. Finally, if vaginal section in the treatment of pyosalpinx is any criterion, the possibility of inadequate drainage through a vaginal opening must be recognized. The writer also apologizes for suggesting that the evacuation and drainage of a secondary pelvic abscess deeply seated in the cul-de-sac, in the absence of any indication of a spreading peritonitis, could be advantageously carried out through the rectum or vagina rather than through a fresh incision in the anterior abdominal wall.

In conclusion, the writer presents this method of drainage of pelvic abscess in full knowledge of the fact that numerous surgical lesions admit of satisfactory treatment in one of several ways, and with no intention to exclude other methods of treatment which in the hands of his colleagues may have yielded equal if not superior results.

DISCUSSION ON THE PAPERS OF DRS. DOWD, OLMSTED, WILLIAM J. MAYO, ROSS, AND ELIOT AND PICKHARDT.

Dr. A. J. Ochsner, Chicago:

For the class of Hirschsprung's disease, which Dr. Dowd has described, I believe that the operation which I described some years ago for bad intestinal stasis cases is especially indicated. It is a two-stage operation, and the first plan is as follows (blackboard demonstration).

You cut off the ileum 10 to 20 cm. from the cecum, carry it up through the abdominal wall through a McBurney incision, insert a large Jacobson catheter directly into the cecum and then attach the other end of the ileum to the colon below the sigmoid flexure. The enlarged colon is then excluded, but at the same time you have the advantage of being able to irrigate this enlarged portion so that the second operation can be done very easily.

In cases like the second case that Dr. Dowd has described the patient could not bear an extensive operation. In a case like that in which I performed the radical operation I have been certain that had I used this method I could have saved the patient. I made use of this method in only one case, in which the patient was given two ounces of castor oil every morning and evening followed by a very large soapsuds enema until the colon was apparently thoroughly empty. She was given two quarts of buttermilk and a pint of cream, together with some fruit-juice, each day, which supplied a sufficient amount of food, so that she looked much better at the time of the operation than at the beginning of the treatment. At the time of the operation she might possibly have borne the excision of the colon, but having had the experience of losing a case which I should not have lost, I chose the above operation. Later I irrigated the intestine daily through the catheter and the patient picked up very rapidly. I believe that this two-step operation will succeed in saving a lot of these patients.

In patients in whom it would be quite impossible to make an anastomosis if operated on before the preliminary treatment had been instituted it is possible to make this anastomosis quite comfortably.

The lantern slides that we have seen show how impossible it would have been to carry the small intestine down below the sigmoid to make an anastomosis.

Dr. William B. Coley, New York:

I should like to discuss Dr. Dowd's paper on Hirschsprung's disease. I had one case similar to Dr. Dowd's third case in which the enormous distention of the colon was probably due to mechanical obstruction.

The patient was about fifty-five years of age, with a history of several times during the preceding two or three years having had almost complete obstruction. The bowels, with the persistent use of cathartics, would be relieved after five or six days. At the time I first saw him he had had no passage whatever for ten days. His abdomen was very greatly distended and he had a temperature of 100° and a pulse of 120. His condition was so serious that he would not have stood an operation of any magnitude.

Under cocain anesthesia I made an incision over the sigmoid and sutured the enormously distended sigmoid to the abdominal wall. I then inserted a trocar which permitted the escape of a large quantity of gas. The following morning I made an incision through the sigmoid through which was evacuated a very large amount of liquid fecal matter. The patient made a very satisfactory recovery, and at the end of three or four days began to pass fecal matter through the rectum; after ten days the stools were normal and the opening in the sigmoid quickly closed. About two weeks later signs of obstruction again developed, with rapidly increasing distention. It then occurred to me that the cause of the obstruction was probably a very movable sigmoid that had become partly distended with gas, resulting in a partial volvulus which, with increasing distention, became complete. To relieve the gas I made another small opening through the old incision and inserted through the lumen of the bowel the end of a rubber catheter, about $1\frac{1}{2}$ inches long, to which was fastened a safety-pin. As soon as the bowel was emptied of gas ordinary movements followed. In order to make the opening a permanent one, at the end of one week I inserted a silver tube $1\frac{1}{2}$ inches long (about the same size as the catheter), with a wide flare to prevent it from entering any farther, and which was held in place by tape passing around the abdomen. At the lower end of this tube was an opening 1 mm. in diameter, just sufficient to relieve the gas without permitting the leakage of fecal matter. The patient was shown before the New York Surgical Society about five years later, twelve years ago, and is alive and well today. During the entire time he has continued very comfortably until the last year, when he again had occasional attacks of distention and considerable leakage about the tube. I think this patient could not possibly have survived any major operation, and I believe the method used in this case to be worthy of trial in similar conditions.

S. J. Mixter, M.D., Boston, Mass.:

These cases of enlarged colon interest me very much, as it has been my fortune or misfortune to have had quite a number of them. An interesting point in regard to them is that a comparatively large

number of them occur in insane asylums and have been operated on or found at autopsy. I have had two such cases; one of the patients was apparently hopelessly insane. He had an acute obstruction from volvulus of an enormously dilated colon and recovered. He soon recovered his mentality and has been doing his work as professor in one of the large universities for many years. Has autointoxication anything to do with the mental condition?

In regard to the two-stage operation, theoretically it is fine and in general works well, but occasionally at the second operation certain adhesions may be found where none existed at the primary operation, and where there were clinically no signs of peritonitis. It is impossible to tell beforehand whether these adhesions will occur after a primary operation or not.

Dr. Willy Meyer, New York:

In my division at the Lenox Hill Hospital we prefer to employ the pararectal incision. It gives always ample access to any condition met inside of the abdomen.

I have frequently seen my adjunct surgeon, Dr. Pickhardt, who is the joint author of this paper with Dr. Eliot, do these operations; he was raised at the Presbyterian Hospital and prefers McBurney's intermuscular incision, and uses it with good success. Yet I believe that if we use the pararectal entrance we can always do whatever may come before us easier than through the intermuscular.

We never irrigate the peritoneal cavity, and in the presence of pus like to place a cigarette drain, with a tube in its center, down into Douglas's pouch; it emerges at the lower angle of the wound. We stitch the wound in such a way that the drainage tube will not slip into the very lowest corner of the wound but rests in a kind of buttonhole, the lower angle of the different layers having been closed by one or two sutures. We treat all these patients afterward with a pronounced right Sims posture. We use Fowler's posture combined with the right Sims for the first three or four days, continuing with a slight Trendelenburg posture.

In case of perforation of a gastric or duodenal ulcer, and accumulation of pus in the lower half of the peritoneal cavity, we make a stab on both sides corresponding to McBurney's point and drain the lumbar regions as well as Douglas's pouch. We then use preferably the abdominal with Fowler's posture the first few days of after-treatment.

In the title of Dr. Eliot's paper it is stated that pelvic abscess associated also with other parts of the large intestine is to be discussed. Such an abscess can eventually be extraperitoneal.

I recall one case of this type: The patient was from out of town with

vague pains in the sigmoid region. He had first been treated by another surgeon for a supposed condition of the left ureter. He stretched the latter, thinking there might be a stricture. Then a barium clysma was administered previous to an *x*-ray examination. From that time on he had more trouble. He came under my care with indefinite diagnosis. By exclusion I gradually came to the conclusion that we had to deal with diverticulitis of the sigmoid. The patient then left the hospital for a while, but soon came back with high fever, and while under my care developed a retroperitoneal abscess with pronounced contraction of the thigh. The abscess was opened and about five days later he developed a retroperitoneal fecal fistula, the greater part of the intestinal contents passing through the wound. A week later he developed a fecal fistula through the bladder. I explained to him that it would be necessary to establish an artificial anus at the cecum or ascending colon, in order to lay the left side field of disease perfectly dry. To my regret he insisted on going home, where another surgeon soon performed the proposed right colostomy. A few days later he suddenly died from pulmonary embolism. Unfortunately there was no autopsy.

Dr. Archibald MacLaren, St. Paul, Minn.:

I feel that perhaps I should apologize to Dr. Eliot for the criticism made in my paper of last year regarding the Presbyterian Hospital reports, especially when I made the statement "that rectal or vaginal drainage had not been used in the 250 reported cases." But this was such a "shining mark," and I could not conceive then, nor do I understand now, why rectal and vaginal drainage had never been necessary in any of these cases. From Dr. Eliot's description I rather believe that he was right in a way in this list of cases. Perhaps Dr. Eliot and myself were talking of a different class of cases: Gynecologic and surgical appendicitis are two very different diseases. It almost seems as though we had more neglected cases of surgical appendicitis in the West than you have in the East. Still, Lincoln Davis sees them in the Massachusetts General and Mathews at the Roosevelt Hospitals.

I am particularly interested in the cases that Maurice Richardson described, where Nature cures a certain number of these neglected cases by causing the abscess to open and discharge into the rectum or into the vagina. Why not assist Nature in these cases and lessen the danger of backfiring or secondary rupture of the abscess, which is so certainly fatal?

I believe that Dr. Eliot takes the proper position that rectal and vaginal drainage are equal and alike; that there is absolutely no

difference between them. I do not quite understand why Dr. Eliot excludes these four cases, which were "not in the hospital a long enough time to be considered." If they died in twelve hours after admission nothing could have saved them. If they lived twenty-four hours perhaps rectal or vaginal drainage might have saved them. If they lived forty-eight hours there was a good chance that some of them might have been saved.

I remember Dr. Stillman reporting a case in which he had been able to effect a cure by rectal section after vaginal section had failed. My plea is that in these bad cases, where on the sixth to the ninth day the patient is complaining of abdominal pain and the temperature is rising, that we should make rectal examinations and know that there is no accumulation of pus in the pelvis. If all of our patients get well there is nothing more to be said; but unfortunately some of our patients die, and these are the cases that we hope to help by rectal or vaginal drainage. There are some few cases so desperately ill when first seen in which opening and draining a pelvic abscess through the rectum, or in the case of a woman through the vagina, is the only thing that can be done. This class, however, should have a laparotomy forty-eight hours after drainage.

Dr. D. F. Jones, Boston, Mass.:

Any life-saving procedure in surgery should not be abandoned, and I believe that drainage of a pelvic abscess through the vagina or rectum, in cases of extensive peritonitis and in very septic patients, is a life-saving procedure. That these cases are not so frequently seen now as formerly is true, but that does not lessen the value of the procedure. Many cases of secondary abscess following extensive peritonitis should be treated by this route. The operation can be done with local anesthesia without disturbing the patient in the least.

In extensive peritonitis cases it is my policy to disturb the abdomen just as little as possible, and when there is pus in the pelvis a cigarette wick is placed into the right side of the pelvis, close along the pelvic wall, disturbing the intestines as little as possible. It is true that occasionally a secondary abscess forms on the left side of the rectum, but it is easily drained by the vagina or rectum. It is probably true that Dr. Eliot's technic of washing out the pelvis in these cases gives fewer secondary abscesses, but the immediate result I believe to be less favorable and the danger of intestinal obstruction is considerably increased.

Dr. J. R. Eastman, Indianapolis, Ind.:

It would be well if there were a more definite understanding as to just what constitutes a Jackson membrane. Nearly every surgeon

has his own original conception of what a Jackson membrane is. If such a Jackson membrane is merely a marked dilatation of the branches of the ileocolic artery and vein, particularly of the peritoneal branches, and with a condition of hyperemia, with teasing apart of cells as in hyperemia induced by Bier's method, with separation of the superficial cell layers of the peritoneum covering the cecum, so that we can with the thumb move a thin gossamer membrane independently of the underlying large intestine—if we have that condition in mind, and think of it as a Jackson membrane, then we are considering a phenomenon which is present in every case of chronic appendicitis. If one watches carefully he will find this Jackson membrane in every well-defined case of chronic appendicits. Some of the older writers, such as Treves and Fitz, had in some respects a better understanding of appendicitis in its chronic form than many of us have today, because they viewed it apparently through a wider arc of vision. They saw not merely the involved appendix, but they saw associated with it colitis and pericolitis, adhesions, stasis, etc., applying the designation perityphlitis to the entire pathology. We know that all of this pathology, including Jackson's membrane, may have its origin in an infection of the appendix and be relieved by removal of the appendix.

As to the origin of these other weblike adhesions about the cecum, I agree with Dr. Ross in one respect and disagree with him in another. Many of these pseudoperitoneums are of embryologic origin, and still they are phenomena of inflammation. We must not forget that we may have infections of the peritoneum in the fetus. As a result of such prenatal pathology there develop during fetal life membranous adhesions and folds about the large intestine which first come into evidence in postnatal life and are discovered during operations.

Douglas Reid, of the University of Cambridge, has demonstrated in the fetus the so-called genitomesenteric fold which corresponds precisely in position to the band of Lane, as demonstrated in postnatal life. Here we have a factor developing during the prenatal existence of the organism which has a great deal of practical interest as a postnatal phenomenon in the form of Lane's band. This genitomesenteric fold may well be due to the dragging down of the loose peritoneum of the mesentery of the terminal ileum by the sex gland in its descent, for like the Lane band it courses from the mesentery of the terminal ileum to the internal abdominal ring.

Dr. Alexis V. Moschcowitz, New York:

In the life of every surgeon the spirit moves him at least once to write a paper on appendicitis. The spirit moved me to write my

paper upon the subject,[1] in which I tabulated 2000 cases of appendicitis, which occurred during the preceding few years at Mount Sinai Hospital, New York. I emphasized particularly the nomenclature of the different varieties and found so many of them that I compared my paper to the well-known "57 varieties." Since that time I have studied a good many thousand more cases, and as a result of the larger experience I have simplified the nomenclature so that, particularly from a viewpoint of prognosis, we now recognize only three varieties.

Group 1 is called "appendicitis." With the exception of the occasional case that dies all these cases get well. When they recover they remain well and have no symptoms whatever referable to their old trouble.

The cases of Group 2 I have nicknamed "rightsiditis." Some of these get well and then they belong to Group 1; some do not and then they belong to Group 3.

The cases of Group 3 I have nicknamed "flivvers," and they never get well. They are the ones who are a source of continuous trouble to the surgeon, as their symptoms continue unabated, and to my mind all are thoroughly incurable.

The difference between Dr. MacLaren and Dr. Eliot is really slight. If I understand Dr. MacLaren rightly he advocates pelvic incision as a preliminary measure before operating for the appendix. Once in a while we find a residual pelvic abscess, and in such cases I have no hesistancy whatever in making a rectal or vaginal incision; these cases usually occur late in the treatment of the appendicitis. As a primary measure we never practice rectal or vaginal incisions.

The treatment as I carry it out differs from Dr. Eliot's method by the fact that I do not irrigate the wound and do not remove the tube. The tube is gradually shortened, and at the end of twelve days is usually removed permanently. My results with this method have been highly satisfactory to me.

Dr. Nelson M. Percy, Chicago, Ill.:

I should like to endorse everything Dr. Mayo has said in reference to pernicious anemia. Notwithstanding all the work that the pathologists and physiologists have done on pernicious anemia, and its relation to the spleen, they have really told us nothing to guide us in the treatment of this disease. We still have to depend upon our clinical observations.

[1] Langenbeck's Arch. f. klin. Chirurg., 82.

The diagnosis of pernicious anemia is not always easy. There is often some question as to the diagnosis, especially in the cases which improve considerably following treatment.

In our work we insist upon five conditions being present before making a diagnosis of pernicious anemia. First, a high color index, together with the presence of a large number of macrocytes, which are the most characteristic features of the blood picture; second, history of remissions; third, an absence of HCl in the stomach; fourth, atrophy of the papillæ of the tongue; fifth, evidence of cord changes if the disease has persisted any length of time.

During the past seven years we have operated upon eighty cases of pernicious anemia and our results have been very similar to those of Dr. Mayo. Our mortality has been a little bit higher: Eight operative deaths, or a mortality of 10 per cent.

In a number of cases in which we simply gave repeated transfusions, combined with dietetic and medicinal treatment, the patients did not do as well on the average as the cases that were transfused and then operated. Every case should be searched for the presence of a focal infection, and when found an effort should be made to remove the focus of infection, not with the idea of removing the cause of the disease but that of simply removing an added burden to an already debilitated patient. The teeth, tonsils and gall-bladder should always be investigated. We found the percentage of infected gall-bladders to be quite high, and in all such cases that were operated the gall-bladder was removed as well as the spleen.

We have been able to trace all but about 10 per cent of our cases, and the results are as follows: Operative mortality, 10 per cent; in 20 per cent the symptoms recurred in so short a time that we felt the operation was not worth while; 30 per cent of the cases were living at the end of three years; 20 per cent between four and five years; 4 cases over six years and 1 case seven years.

None of these cases have been entirely relieved of their symptoms of pernicious anemia. All of them still have the disease, and probably will have as long as they live.

It is evident that removal of the spleen cannot cure pernicious anemia unless we can in addition find the primary focus of infection and remove this. We still believe, however, that in many cases they should be considered surgical. Their surgical treatment should consist of repeated transfusions, the study of and eradication of all focal infections; this to be followed by laparotomy for removal of the spleen and other infected organs in the abdomen, which usually means the gall-bladder and the appendix.

Dr. Frederic N. G. Starr, Toronto, Canada:

This has been a most interesting discussion. I think perhaps I am in the same position as the other Fellows of the Association, namely, that there is more about the large intestine that I do not know than that I do.

We had a curious patient in Toronto some years ago, but I have not heard of her for five or six years. She may have ceased to exist since or during the war. She had sometimes three and sometimes four bowel evacuations in the year, never more than four, yet her health was comparatively good—her skin was like a piece of leather.

On one occasion she had the bowels thoroughly cleaned out and then she went to the old country. She traveled in England and on the continent, and after spending three months returned to New York, where she had her next evacuation of the bowels. Why that woman did not have all the ills that the large bowel is supposed to create is a wonder to me, but instead she had apparently good health.

I was interested in the paper on Hirschsprung's disease, and particularly interested in the remarks of Dr. Mixter on the advantage of not doing a two-stage operation because of the difficulties that one meets when adhesions have occurred. I had precisely a similar experience some years ago and very nearly killed my patient with the second operation, and I think her health has never been first-class since then. On the other hand, in some of the cases in which I have done the one-stage operation it has been very simple and very quickly done.

In the paper of Dr. Ross on adhesions about the cecum there is a condition in the cecum that produces the same symptoms that seem to occur as a result of adhesions in which there is practically no adhesion whatever. That condition is "atony," and I think, as was pointed out in a very large proportion of chronic appendicitis cases in which the appendix was removed, they were in the first place cases of "atonic cecum" associated with an incompetent ileocecal valve.

In a case seen recently, by means of a barium enema we succeeded in getting a negative, by the *x*-ray, showing the duodenal cap, and in another one, two weeks ago, I think if we had persisted we would have got the duodenal cap. We followed the barium as high as the duodenojejunal junction, when the patient complained of great nausea, and we desisted.

In resection of the bowel, since September or October, I do not remember, in the last thirty-six cases of resection of the cecum and ascending colon we have done an end-to-end anastomosis of the ileum to the hepatic flexure of the colon, and in order to obviate the possible partial obstruction that results from the edema at the point of anastomosis I have introduced a short piece of rubber tubing and made

the anastomosis around it. The rubber tube comes away in five days at the shortest time, or seventeen days at the longest time. We are beginning to feel that resection of that portion of the bowel has quite as comfortable a convalescence as an ordinary appendicitis case. There is practically no shock and there has been very little discomfort, and as to the results, we are just getting word from our patients now, who are far enough advanced, and they are reporting themselves better than they have been for many years. One patient reported a year after the operation that she has not been as well for eleven years.

Dr. Charles N. Dowd, New York (in closing):

There are many advantages in the procedure which Dr. Ochsner advocates. If there is a way of securing anastomosis without the use of a temporary stoma, of course it is an advantage to do so. The conditions must differ with the individual cases, but as more and more surgery of this kind is done, the proportion of emergency cases will diminish and there will be some patients in whom a suitable anastomosis to the sigmoid can be made without the use of the stoma. At the present time, however, most of these operations are done with a foul colon, and a method which guards against sepsis is very desirable.

The prevention of adhesions is also very important, and for this purpose it is desirable that the greater portion of the abdominal work should be done at the primary operation. No doubt the provision for leakage will sometimes be made by a vent proximal to the anastomosis, and sometimes by a double-barreled stoma at the anastomosis; but, of course, the procedure must differ with individual cases.

Dr. Coley's case is included in the tabulation. The result was surely brilliant.

Dr. Ingersoll Olmsted, Hamilton, Ontario, Canada (in closing), showed the patient.

Dr. William J. Mayo, Rochester, Minn. (in closing):

I believe Dr. Charles H. Mayo was the first to use the term "Lane's kink." In my experience Lane's kink seldom gives rise to trouble unless operated upon, following which trouble may ensue. Certain queer metabolic disturbances take place in the right half of the large intestine from causes we do not understand. We have made a number of right-sided resections of the large intestine with some good results. Nearly all the patients we have operated on had had from one to seventeen operations; we have never felt sure whether we benefited the patients because we had relieved primary conditions or because we had relieved them from the results of the various operations they had

undergone. In Lane's disease there is too little pathology and in Hirschsprung's disease too much.

With regard to questions of mortality: I always want to know whether the discussion concerns the number of operations or the number of patients. For instance, one patient may have five operations; if he dies the operative mortality is only 20 per cent, but the patient is just as dead as if the mortality had been 100 per cent. Multiplying operations improves percentages but does not necessarily mean fewer deaths.

I agree with Dr. Dowd that in excision of the large intestine the two-stage Mikulicz operation is often the best procedure. In handling an obstructed large intestine a distinct fecal odor is often noticed on the gloves. One need not be surprised, therefore, to have an enormous amount of adhesions following such operations. If the adhesions can be made to form in a manner to prevent kinking and secondary obstruction they may be made useful. Dr. Balfour has shown that this can be accomplished by passing a heavy rubber stomach tube through the rectum and well up into the descending colon, or up into the small intestine if an ileocolostomy has been made, the tube extending high enough to form an easy curve. This acts like a form for cement. The adhesive material, the result of the unavoidable infection, becomes useful in maintaining such easy curves and preventing subsequent angulation. This procedure is especially valuable in preventing the rather frequent incomplete obstructions of the lower sigmoid at the promontory of the sacrum, so forcibly pointed out by Handley in his paper on "Ileus Duplex."

Dr. George G. Ross, Philadelphia (in closing):

As a matter of fact there are some thirty odd cases on record in which the evidence of prenatal peritonitis existed.

As to Dr. Starr's reference to an atonic cecum, I have seen it as a frequent occurrence. I have likened it to a stopped drainage system and have called it a cecal cesspool, a name which gives a word picture of the condition.

Dr. Ellsworth Eliot, Jr., New York (in closing):

In the first place abscess in the pelvis of appendicular origin is so much more frequent than abscess associated with infection in other parts of the large intestine that while there is no essential difference in either its physical signs or treatment it seemed best to exclude the consideration of the much rarer variety from the limits of this paper.

In the second place I repeat and emphasize that the incision, irrespective of its type, should be of adequate extent so as to permit

the proper exposure and treatment of the appendix. Although it has been our custom to use the intermuscular incision, yet the pararectal incision may be equally advantageous. (I think, however, that the intermuscular incision does less damage to the abdominal wall than other methods of approach, and that after its use a ventral hernia less frequently develops.)

In the third place I should like to emphasize the advantages of the semirecumbent posture, which is adopted in all cases as soon as the effects of the anesthetic have passed away.

The operative mortality, which I did not have time to discuss but which will be included in the published paper, has steadily decreased during this period of twenty years. This I think may be ascribed to the fact that in our section patients seek aid very much more promptly now than they did twenty years ago. Some credit also should be given to improved operative technic and to the present type of drainage.

In regard to the question of a counteropening through the rectum or vagina, one must be careful to distinguish between such an opening made at the primary operation and one made as a postoperative measure to relieve defective drainage or to promote drainage of a secondary abscess during the period of convalescence. To demonstrate that such a counteropening at the primary operation is unnecessary has been the chief object of this paper. As a secondary measure, however, for the drainage of a deeply seated abscess in the pelvis, such a counteropening, I believe, would prove of great value. There has been no occasion for its use, however, in any of the cases herewith reported, with the exception, possibly, of the two instances of defective drainage after the use of the Mikulicz tampon, which occurred some twenty years ago.

Irrigation of the abscess cavity at the time of the primary operation I consider desirable; it can be quickly done and removes a considerable amount of foul material which might easily form the basis of a secondary abscess. Perhaps it is not too much to suggest that lack of irrigation on the part of those who do not approve of its use may account for the occasional development of a secondary pelvic abscess.

AN OPERATION FOR POSTOPERATIVE VENTRAL HERNIAS

By ARTHUR DEAN BEVAN, M.D.
CHICAGO, ILL.

During the last three or four years we have had on our surgical service a number of large postoperative hernias. We have studied these cases with special care in the hope that we could develop a technic which would be an improvement on anything we had before employed. As a result we have been able to develop a technic which has given very satisfactory results. I believe that it is worth while presenting to the Fellows of the Association as a practical improvement of definite value. The principles of the operation were largely worked out in another group of cases. I will therefore take enough time to relate to you the way in which we happened to develop this particular plan.

In the course of a series of years we have had in our clinic a number of cases in which after laparotomy, either with or without infection, the laparotomy incision opened up after the stitches had been removed at the end of nine or ten days. These cases usually occurred in individuals of low vitality in whom the process of wound-healing was especially slow, in cases of individuals who had been starved by long-standing pyloric obstruction, either from carcinoma or ulcer, or a few cases of congenital pyloric stenosis done in infants in advanced stages of starvation on account of the obstruction, and in a few cases of operation on the bile tracts, either from infection or carcinoma, where wound-healing was slow on account of the

bad condition of the patient, or because of the infection extending from an infected gall-bladder or bile tract to the operative incision. Before the introduction of local anesthesia these cases presented a much more difficult problem than they do now, because when one of these wounds opened up and the omentum and abdominal contents pushed their way out it was often a serious matter to give the patient a general anesthetic, because sometimes in taking a general anesthetic further evisceration occurs during the stage of excitement, which further complicates the case. After the introduction of local anesthesia we soon learned to handle these cases by anesthetizing the edges of the incision sufficiently widely so that we could then introduce our through-and-through sutures of silkworm gut, and we frequently tied these sutures over a piece of iodoform gauze or a piece of rubber tubing or a short piece of cigarette drain so as to make it possible to leave them in position longer than an ordinary suture. Although the use of a rubber drain or piece of gauze was an improvement over the simple suture it was not entirely satisfactory, and I then began, some years ago, to use large buttons about an inch in diameter, an ordinary pearl or porcelain button, and that has answered the purpose very much better than any other mechanical contrivance that we could find. In a considerable number of cases we use these buttons with great satisfaction, frequently leaving the buttons *in situ* for two weeks or more. This was often necessary because where an accidental opening up of the wound occurred the resulting wound-healing was more or less complicated with infection or suppuration. We then took another step forward in this work. We began to use these button sutures at the time of the original operation in the case of handicapped patients in whom it seemed possible that such an accident might occur. In the case of a resection of the stomach for carcinoma or a gastroenterostomy, or a badly infected bile-tract operation, we would put in about three of these button sutures at the time of the original operation in addition to the regular scheme of suturing which is usually employed, and would then remove

the skin sutures and the ordinary silkworm-gut sutures, which were placed between the buttons at the usual time of eight, nine or ten days and leave the button sutures for a longer period until we were sure that wound-healing was complete, leaving the buttons in anywhere from twelve to sixteen or eighteen days, and in some cases even longer. We have felt that this matter was really a very important thing in our work, as it has completely eliminated the occurrence of the opening up of any of the laparotomy wounds since I introduced it on our service.

Of course we make no claim of originality in this work. The method is essentially the same as the old quill suture which has been used for centuries, and many operators have used lead plates, and used, as we did for a long time, rubber tubing or pieces of gauze over which to tie these large fixation sutures. The fact remains, however, that the ordinary button to be found in the shops proves to be in surgery the best mechanical device that can be employed, just as it is in buttoning up one's coat; and simple as it is the button has stood the test of time and of universal use. All surgeons of any considerable experience have had repeatedly the tragedy of the opening up of these abdominal wounds and dread the occurrence of this accident, because it not infrequently leads to a fatal termination, and because it is so liable to result in a weak scar with resultant postoperative hernia. Any plan, therefore, that will eliminate these serious accidents, no matter how simple, is worthy of serious consideration and of general adoption. It is because of that fact that I do not hesitate to present this subject at a meeting of this Association on the basis of the experience of our service and to urge the thorough trial of this plan, which I feel will lead to its general adoption.

As time has gone on we have been so much impressed with the value of the button-fixation sutures in the abdominal work that we are now employing them in a very considerable percentage of our laparotomies. They have advantages even in perfectly clean, simple cases, and in individuals who are good surgical risks, and in whom normal wound-healing occurs in

the usual time of eight to ten days. They permit of the early removal of the silk skin sutures and of the simple silkworm-gut sutures—that is, removing on the fifth or sixth day of the skin sutures and about the seventh day of the ordinary silk sutures, leaving the buttons to a later period. In removing the buttons, for instance, we have three or more button sutures, we frequently remove them by instalments, that is, one at a time, very frequently removing two or three on, say, the twelfth day, leaving a single button suture until the fourteenth or sixteenth day, when there is no further indication for its use.

It came about in a very natural way that after we had developed this button technic that we applied it to operations for postoperative hernias in abdominal incisions, because frequently in these cases it is desirable and necessary to maintain a coaptation of the structures for a longer period than in an ordinary primary clean laparotomy incision. We began using the button sutures in cases of large umbilical hernias, in which we employed the Mayo method of imbrication from above downward, and obtained really great satisfaction from their use. One of our early cases of postoperative hernia was in a woman, who weighed about 250 pounds, who had a very large postoperative hernia of a large incision which split the right rectus muscle. This hernia was about the size of an ordinary head (Fig. 4). It had never been associated with any obstructive symptoms, but it gave her very great discomfort, and it was impossible to maintain reduction by any abdominal support or truss which she could secure. She had a bad heart, and on that account I did not want to give her a general anesthetic. Her condition had become such a handicap that it was difficult for her to get about with any degree of comfort, and operative relief seemed to be very desirable if it could secure cure or improvement of her condition. Under local anesthesia I infiltrated the whole line of the scar and made the incision; about twelve to fourteen inches long, dissected out the old scar and isolated the huge hernial sac. I then conceived the idea that inasmuch as she had no obstructive symptoms, and never had had any,

of not opening the peritoneal cavity but of invaginating the sac with several rows of medium-sized catgut sutures. It required three or four of these layers to return the sac completely into the abdominal cavity. As we did this we gradually drew together the aponeurosis of the abdominal muscles, which formed the anterior sheath of the rectus. The invagination is well shown in Fig. 1, which represents the first two layers of invaginating catgut sutures. The result of the invagination is well shown in Fig. 2. This, of course, is diagrammatic; the

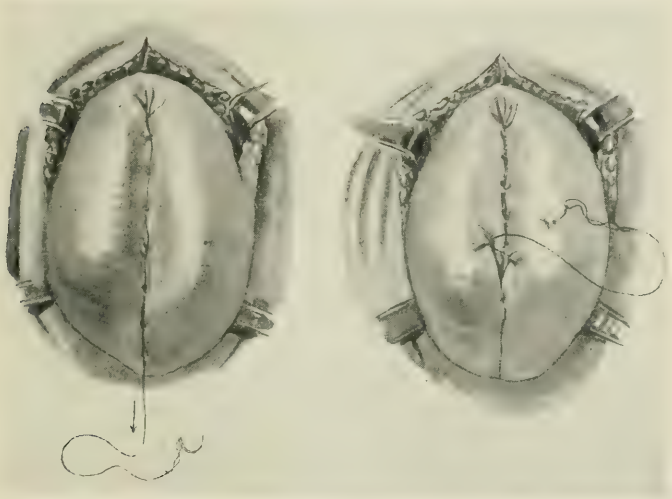

FIG. 1

dotted lines show the hernial sac before invagination and the hernial sac is shown after invagination into the abdominal cavity. The figure also shows a cross-section of a single button suture which passes through the skin, superficial fascia and anterior sheath of the rectus and rectus muscle on both sides. When this is drawn together it continues to invaginate by bringing A to A' and gives us the same effect as the imbrication such as we use in the Mayo umbilical hernia operation. We were compelled to use in that case a large number of button

sutures, I think six or eight, as shown in Fig. 3. In this plate you will also see a single button suture with a double thread passing through the needle. Fig. 4 shows a sketch of a huge ventral hernia and the line of incision which we employed. Fig. 5 shows the dissection of the peritoneal sac.

Fig. 2

Much to our gratification this woman had little difficulty after operation. The huge wound wept a good deal of primary wound secretion, but no suppuration developed. We removed the skin sutures at the end of six or seven days and began at about the twelfth day to remove the buttons, first removing every

other one, leaving but three button sutures; then on about the sixteenth day we removed two of the remaining three and left

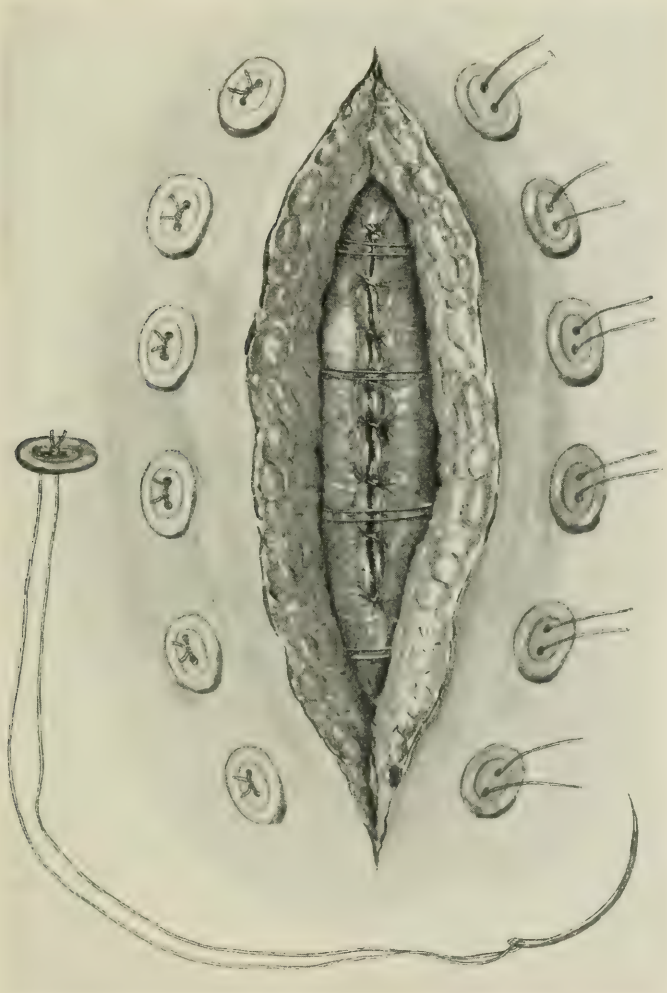

FIG. 3

one of the center button sutures until about the twentieth day. On account of the primary wound secretion we dressed the

wound with moist boric dressing every day, which seemed to be of real service in the case. The result has been very satisfactory. She is cured of the hernia. She wears a large, loosely fitting

FIGS. 4 and 5

corset which seems to give her more comfort than any form of abdominal support (I mean the ordinary woman's corset). She is now able to do her own housework, and she is very grateful for her cure.

I am describing this case somewhat in detail because the hernia was a huge affair, and it was among our early cases, which convinced us of the value of this technic. Since that time we have used it in a large number of cases, and in all but one or two cases we have been able to cure the hernias completely and obtain practically a normal abdominal wall.

In hernias which have never had any obstructive symptoms, especially those that have been retained in position by trusses without any obstructive symptoms, we have felt warranted in doing this type of operation without opening the peritoneal cavity. In hernias which have had obstructive symptoms, of course, it is much wiser to open the peritoneal sac to free the adhesions and relieve, as far as we can, any cause of obstruction before we complete the invagination.

One wants to use good-sized buttons. I think it makes little difference what material is employed, whether it is metal or porcelain or the ordinary shell-pearl button that is so commonly found in the shops. We use more frequently a button about seven-eighths of an inch in diameter. We have, however, some smaller buttons, one-half and one-third of an inch in diameter, and have used the smaller buttons especially in cases of operation for congenital pyloric stenosis. Any of these buttons can be very readily sterilized by boiling or even by keeping them in a jar of alcohol, which I think answers the purpose and makes them quite safe. We have used both silkworm-gut and silk for suture material. I prefer silkworm-gut to silk, for I think there is less likelihood of cutting.

I might briefly describe to you the technic of using these buttons in the ordinary laparotomy incision, as shown in Fig. 6. The upper portion of this plate shows the laparotomy wound closed in the usual way by catgut through the peritoneum, then catgut through the anterior sheath of the rectus, then silkworm-gut sutures outside of the peritoneum, and then three-button sutures.

The lower part shows a cross-section at the point of introduction of one of these buttons. You will note that the buttons

are put well back from the line of incision, probably an inch and a half from the edge. It is well not to put the button too close to the incision. The suture passes through the skin, superficial fascia, anterior sheath of the rectus and rectus muscle

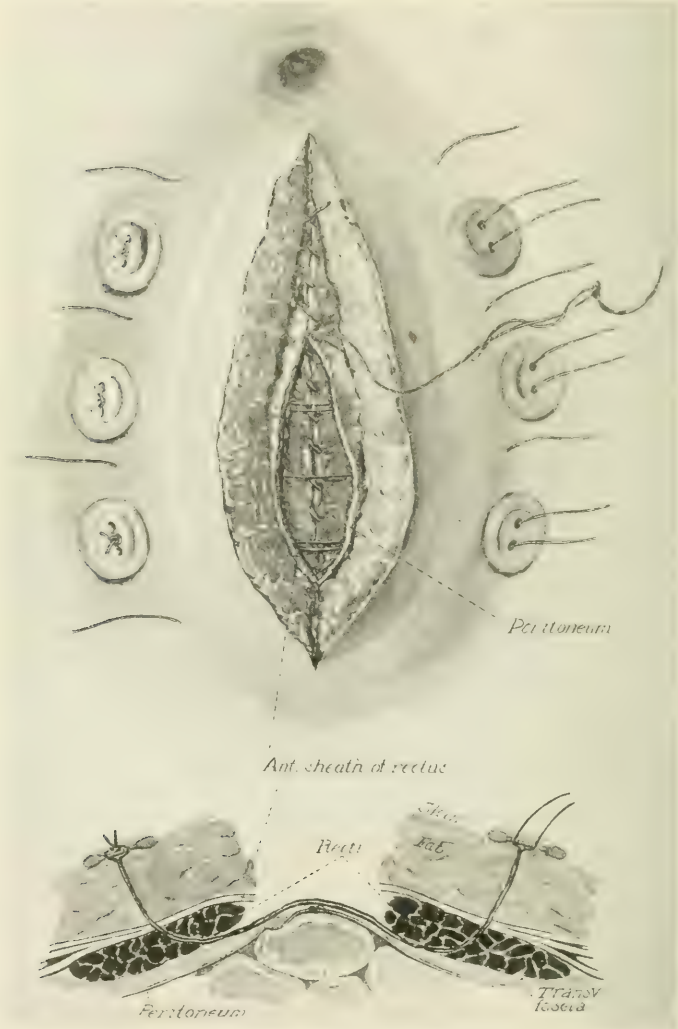

FIG. 6

and all the tissues except the closed peritoneum. In adopting this technic it is well to make a complete closure of the wound before tying the buttons, that is, in addition to closing the peritoneum and the anterior sheath of the rectus with catgut, tie the ordinary silkworm-gut sutures and introduce the black-silk sutures through the skin before tying the button. The button should not be tied tightly. The purpose is to maintain a simple, firm and even fixation without wrinkling of the tissues.

In closing I want to again emphasize the importance of this simple method. Many of the most valuable things in surgery are very simple. From our experience now for a number of years I want to testify to the great value of these button sutures in abdominal work and urge their adoption. I might add another word in regard to their special value in colostomy, where we are using a single button suture to bring up the sigmoid or the cecum. I frequently make the colostomy with a single suture with the addition of a few interrupted skin sutures. Here, of course, the button suture passes not only through the layers of the abdominal wall, but also transfixes the mesentery of the gut and acts as a bridge, dividing the proximal from the distal portion of the intestine. The button in the colostomy cases has the advantage of enabling us to maintain the suture in position for a considerable period if desired, frequently as long as twelve to fifteen days.

DISCUSSION

Dr. Alexander Primrose, Toronto, Canada:

I should like to refer to another button other than the one which Dr. Bevan describes. In these cases of ventral hernia we do not sufficiently utilize the strength of the superficial fascia. The deep layer of the superficial fascia of the abdominal wall is very strong, particularly below the navel, and should always be employed in such fashion as to secure its support in addition to that obtained from aponeurosis, muscle, etc.

Some years ago I was impressed, when visiting the clinics in Paris, by seeing Tuffier perform an operation which he styled "the manufacture of an abdominal bélt from the tissues of the abdominal wall." The operation is quite simple and I have employed it with success in more than one instance. Dr. Bevan referred to patients who weighed as much as 250 pounds. It is in these very obese patients that Tuffier's operation is of value. We all are familiar with the operation of removing a large section of the skin and fat from the abdominal wall in such fat subjects. The section is oval in shape and runs transversely across the abdomen. Tuffier's operation is an ingenious modification of this method. The operation is begun by making a circular cut around the umbilicus, about half an inch from that structure. This is carried down to the aponeurosis, dividing the skin and both layers of the superficial fascia. The fat, of course, is mostly carried in the interstices of the superficial layer of the superficial fascia, and the deep layer of the superficial fascia remains as a membranous, well-developed structure. By the circular cut around the umbilicus that structure is isolated and stands out, surrounded by a pillar of fat and fascia about an inch and a half in diameter. The height of the pillar depends upon the thickness of the fat and fascia. The oval section of skin and fascia to be removed is now mapped out in the manner usually employed in such obese cases, but the upper transverse cut passes, say, two inches above the umbilicus and the lower incision as far down as may be necessary, for our purpose, in the hypogastric area. This section of skin and fat is now removed entirely, as in the operation usually employed in such cases. Beneath the section thus removed the abdominal aponeurosis remains bare over the entire area, and in the upper portion of that area the isolated umbilicus stands out surrounded by its pillar of fat and fascia. Attention is now directed to the skin and fat of the hypogastric area below the line of the lower transverse incision. This area is undermined, the cutting being done between the deep layer of the superficial fascia and the aponeurosis. In this way a flap is isolated, consisting of the skin and fat of the

hypogastric area. This, of course, includes the deep layer of the superficial fascia. A button-hole is now made in the flap about an inch and a half below its upper margin. This button-hole is made sufficiently large to receive the pillar of fat and fascia about the umbilicus. The lower flap is now brought up and buttoned on to the isolated umbilicus, and sutures are introduced to retain it there. The operation is completed by introducing sutures across the abdomen in such fashion as to close the wound entirely from side to side.

This operation is of distinct value as an adjunct to the operative procedures carried out upon the aponeurosis and muscle. I am not discussing these procedures, and the suggestions of Dr. Bevan in that regard are of great value. The operation of Tuffier, however, may well supplement such procedures, and in my own experience has proved of very definite value.

Dr. Archibald MacLaren, St. Paul, Minn.:

I am particularly interested in Dr. Bevan's reference to the use of local anesthesia, as it recalls to my mind an experience which I had several years ago with a patient who had a very bad ventral hernia that had been operated upon three times, and each time was followed by a prompt recurrence, the stitches evidently being broken by the vomiting after a general anesthetic. In our own experience once in every 300 laparotomies the abdominal wound is either coughed or vomited open. The coughing and vomiting in these cases is to a great extent done away with by the use of local anesthesia.

The procedure that Dr. Bevan has suggested I think is an excellent one, and I will certainly try it.

Dr. William B. Coley, New York:

I regard the technic of Dr. Bevan as excellent, and if we could always be sure of not finding bowel or omentum adherent to the sac I think the method would be a very efficient one to employ.

I raise the question whether we could always be sure that there were no adhesions. In my own experience adhesions have been the rule, and I am fearful that in inverting and suturing the sac in the way described the needle might catch up a portion of the adherent gut or change the position of the adhesions in such a way as to provoke obstruction later on. However, in the absence of adhesions I believe it might be a valuable method. Whether it would give any better results than the Mayo method of overlapping, time alone would tell. I believe the button to be an admirable step in the technic, and useful in most abdominal operations in which there is likely to be tension.

Dr. Arthur Dean Bevan, Chicago (in closing):

In closing I have nothing further to add except one point referred to that I think should be emphasized, and that is that the use of some more secure means of closure than we ordinarily use might prevent a great many of these hernias. I think that the point that Dr. MacLaren has made is well taken, that many of these patients cough and vomit and partly open up the wound, and partial opening of the wound without being completely open is the cause of the postoperative hernia.

My conception is that if we have a more efficient means of maintaining approximation of our surfaces such as this button affords, or some similar means, that we would prevent very often the formation of these hernias. I know myself that I have felt very much more comfortable in handling a good many of these wounds, especially of the upper abdomen, since we have been employing this button. When the patient coughs or vomits, if these buttons are properly applied I feel quite comfortable that the patient will do no damage. Often in operating on some of the patients when they would feel like coughing I would say, "Go ahead and cough."

In a practical way no scheme that we have developed in our service has given us quite as much satisfaction as the use of this very simple device.

DEATH AND CREMATION: SOME PERSONAL EXPERIENCES OF ONE OF YOUR COLLEAGUES

By W. W. KEEN, M.D.
PHILADELPHIA

I.—Soon after December 31, 1871, when we moved into 1729 Chestnut Street, my present residence, a neighboring undertaker (evidently appreciating my abilities more highly than the general public seemed to do, as shown by my visiting book) called upon me, and, with some embarrassment, requested the "privilege of leaving some of his professional cards on the table in my waiting room." Doubtless it was his desire to cheer up my infrequent and desponding patients by the assurance of appropriate care in preparing for and conducting the expected, or the at-least possible, obsequies, if I were the attending surgeon. I felt compelled, however, in the interest of my reputation, to say a gentle "No!"

II.—A few days after the celebration of my eighty-fourth birthday, I received a beautiful circular, with fine half-tone illustrations and an enticing invitation, which, in view of my age, seemed most appropriate. It was a folder headed, "The ———— Burial Company." The half-tone cuts were initiated by a lovely auto-hearse. The rear double doors were wide open, disclosing a cool, clean and inviting interior, while on each side stood a suitably garbed attendant, politely suggesting that I be carried in, for, *ex natura rei*, as my old professor of mathematics

at Brown used to say, it would be quite impossible for me to enter by my own motion.

I could not refrain from replying to such a kind proposition. I thanked the company for their gentle hint that if I was not already dead, very surely I soon ought to be, "but," I added, "as I have other plans in view at present, I must decline with thanks."

III.—Shortly afterward, Act III of this drama was sent me, and, strange to say, as an "Ad" in the *Weekly Roster* of medical meetings in Philadelphia. The *Roster* was pleading for "Our Advertisers: their Products and Services."

The first "service" was a commendation of cremation. They mentioned in alphabetical order (not, be it observed, in chronological order) of cremation the names of "some of those in the profession who expressed their request and *whose desires to be cremated have been fulfilled* at the Crematory," etc. In the list of those who had been declared officially dead and cremated I found my own name.

By the way, in view of such undue celerity, who can now call Philadelphia "slow"?

I pinched myself to find out whether I consisted wholly of ambulant ashes. I seemed to be real flesh and blood—but there it stood, my "desire to be cremated had been fulfilled." However, I concluded to eat and drink and tomorrow—or at some later date—I would be cremated again.

IV.—But the report of my death would not down. As if "to make assurance double sure," two or three days later still, I received the annual report of a religious charity, and, among the annual donors of a modest sum for its support, there was my name, preceded by a "star," which a foot-note explained meant "deceased."

Being a religious charity, of course no false statement could find a place in its reports. I *must*, therefore, be at least theoretically *dead*. At least there is one incidental advantage

of theoretical decease, viz., that the said religious charity could hardly expect any further annual "checks" from a disembodied spirit.

To reassure you—for I am unable to be present in the flesh at this session of the Association—I refer you to my antivivisection friends. They will be compelled, reluctantly of course, to confess that I was certainly alive a month ago, for at that time I had stirred them up a bit by a letter published in the *Boston Medical and Surgical Journal* of May 5, and in the Philadelphia *North American* of May 8, 1921, which I hope you will not fail to read.

If you insist on a certificate, sworn to before a notary public by my family physician, that I am still alive, I will gladly furnish one.

TRAUMATIC PANCREATITIS[1]

By H. BEECKMAN DELATOUR, M.D.
BROOKLYN, N. Y.

Abdominal traumatisms are accompanied by injury to the pancreas in only a very small proportion of cases. Stuart[1] gives a review of the literature to date with a bibliography of 54 cases, including 1 of his own. This list does not contain cases of perforating wounds in which the pancreas was injured. It is probable that the cases reported do not include all in which the pancreas was involved. Some have either not been reported or have not been recognized.

In thirty years' continuous hospital experience in two hospitals, where the daily number of severe accidents is large, the case here reported is the first in which we have observed injury to the pancreas.

In some of the more severe cases of abdominal injury in which death followed in a few hours, and neither operation nor autopsy was performed, pancreatic injury may have been present. In the cases operated involvement of the pancreas was not observed, and in the nonoperated cases the symptoms were not suggestive of this condition.

Penetrating abdominal wounds are not frequently accompanied by injury of the pancreas, according to published statistics. Surgeon-General Wallace[2] gives only 5 cases of injury of the pancreas in a total of 965 penetrating abdominal wounds reported, and in 300 cases collected by Fraser and Drummond[3] only 1 involved the pancreas.

[1] Read by title
[2] Northwest Medicine, March, 1921.
[3] Lancet, 1917.
[4] British Med. Jour., March, 1917.

CASE.—Subcutaneous laceration of the head of the pancreas; operation. Pancreatic pseudocyst; operation; recovery. C. S., schoolboy, aged thirteen years, admitted to St. John's Hospital, Brooklyn, November 21, 1919, at 1.20 P.M. Boy was knocked down by a wagon, the rear wheel of which passed diagonally over the body from the right thigh upward, and to the left across the body to the lower left ribs. There was moderate shock and complaint of pain in the upper right thigh and also in epigastric region.

Physical Examination. Well-developed boy, with slight skin abrasion at the upper part of right thigh and slight ecchymosis across the abdomen. Abdomen: No distention, no muscular rigidity, moderate tenderness on pressure in epigastric region. No dulness in either flank; both testicles were drawn up in inguinal canal; no symptoms of internal hemorrhage; shock slight; urine analysis negative, no blood present. Just after admission vomited a large amount of undigested food; following this pain was much relieved. Temperature 98.6° F.; pulse, 80; respiration, 30.

During the first twelve hours the patient complained at intervals of severe pain. Vomited a bile-stained fluid at frequent intervals. After each attack of vomiting pain would disappear.

During first eighteen hours, hourly records gave pulse 92 to 96; temperature, 99° F., and respirations 32. During this period the only symptoms of note were the persistent vomiting and recurrent attacks of quite severe pain. The question of internal injury was carefully considered, but with so little shock and no evidence of hemorrhage it was considered best to carefully watch the symptoms before making an exploration.

The following morning the patient was seen by me. There was no abdominal distention, moderate rigidity of the recti above the umbilicus and tenderness in epigastrium; there was evidence of fluid in the abdominal cavity. Temperature, 100.5° F.; pulse, 100. Vomiting still continued at short intervals. It was evident that there was some abdominal injury. Exploration advised. A tentative diagnosis was made of either rupture

of the liver with moderate hemorrhage, or contusion or laceration of the mesentery with hemorrhage.

Operation, twenty-four hours after receipt of injury. Incision to right of median line in upper abdomen. On incision of the peritoneum there was an escape of considerable bloodstained fluid and a few blood clots. There was no evident injury to small or large intestine, spleen or liver. In the region of the duodenum the entire area was very edematous and bloodstained. On further exploration the head and about one-half of remainder of the pancreas were found lacerated and contused. The pancreas, through the lesser omentum, was exposed, and three iodoform cigarette drains carried down to the injured tissue. The drains were brought out through the original incision and the wound closed in layers.

During the exploration very extensive areas of fat necrosis were observed in the omentum. Although the operation was not a prolonged one, and there was no excessive bleeding, the shock following was very marked. The pulse became 136–150 and very weak, and so continued for twenty-four hours. The temperature the first night reached 103° F., and the following noon 103.8° F.; pulse, 130; respirations, 40. There was extreme restlessness and thirst. A profuse sanguineous discharge from the drains was extremely irritating to the skin.

November 24. Much less restless and thirst has disappeared. Abdomen slightly distended; moderate amount of pain and no vomiting since operation.

November 26. Temperature 99.8° F.; pulse, 100. General condition good. Has vomited once—milk stained with bile.

November 29. Has been complaining of hunger. Given soft diet, which was taken with relish. Normal bowel movement. Temperature, 99°; pulse, 80. Iodoform drains removed. Much irritation of skin about wound.

December 3. Very little discharge, all drains removed. Complains of pains in epigastrium, especially at night. Is very restless and vomited greenish fluid during the day.

December 9. During the past days has complained a good

deal of pains in the epigastrium, especially at night and after eating. Abdomen slightly distended, particularly in the epigastrium. Enema given with satisfactory result but without relief of pain. Has vomited at intervals. Is very irritable and restless.

December 10. Temperature suddenly rose to 102.4° F. Patient was very restless and vomited twice during the day. No local abdominal findings. Partial obstruction of bowels by adhesions was suspected.

December 23. Since last note there has been some improvement in the general condition, vomiting only occasionally, and pain usually quite severe each night. Has been out of bed part of each day during the past week. Has felt better and is hungry. There have been occasional attacks of abdominal pain but not as severe. Has vomited only once or twice. Today, following an attack of intense abdominal pain, the temperature suddenly went to 102.6° F. There was some abdominal distention and tenderness over pancreatic region.

January 4, 1920. Since last note the temperature has continued between 99° and 101° F. He has been very comfortable during the day, but during the nights has had severe attacks of pain that cause him to cry out. During the past two days a swelling has been evident in the epigastric region and has enlarged rapidly. Blood count: leukocytes, 27,000; polymorphonuclears, 85 per cent. Vomited a large quantity of dark-green fluid once each day for three days.

January 5. Leukocytes, 21,000. Mass in upper abdomen much larger. Pains still continue. Diagnosis of pancreatic cyst made and operation advised.

January 6. *Operation.* Incision made in back just below twelfth rib, two inches to the right of spine. Through this the posterior abdominal wall was opened and the mass was found just internal to the upper pole of the kidney. This was incised and about three pints of clear fluid evacuated. A drainage tube was inserted and wound closed about it.

January 8. Patient's condition improved immediately after

the operation. Pain and vomiting have ceased. There has been a profuse discharge through the drainage tube.

January 19. Marked improvement in general condition. Discharge continues. Tube removed.

January 23. Out of bed. No further drainage. There has been a marked increase in weight during the past week.

February 6. Discharged from hospital.

May 1. General health continued to improve. There has been no recurrence of the symptoms.

The points to be noted in this case are:

1. The comparative absence of shock, although over one-half of the pancreas was badly contused and lacerated.

2. The principal symptoms, both during the first hours after the injury and during the stage of development of the cyst, were vomiting and severe upper abdominal pain, occurring at intervals. In each instance these were immediately relieved by operation. Emaciation was marked until after the cyst was drained, although the boy was eating well most of the time.

3. The ease of approach from behind and the better drainage obtained.

Stuart[1] reports a case of subcutaneous injury to the pancreas due to a direct blow on the upper abdomen. In this case dyspnea was a very marked symptom. Persistent vomiting and pain were also present.

R. A. Stoney[2] reports a case of pancreatic pseudocyst following a shell wound of the abdomen ten months before. Complete recovery after drainage.

Morley[3] reports the case of a private hit in the back by shell fragment July 24, 1916. There was no evidence of peritonitis, but severe aching pain in the epigastrium. Two weeks later a swelling appeared in the epigastrium, with rapid loss of flesh. Temperature, 99.4° F.; pulse, 100. Three weeks after injury operation was performed through an anterior incision, exposing a fluctuating tumor in the lesser sac. The anterior wound was closed and

[1] Loc. cit.

[2] British Med. Jour., April 13, 1918.

[3] Ibid., March, 1918.

the cyst opened by an incision from behind below the twelfth rib on the left. About four pints of clear fluid evacuated. Recovery slow.

C. A. Roeder[1] states that in 47 cases of pancreatic cyst 20 per cent gave a history of abdominal trauma.

The pancreas is occasionally injured during operation on other organs. While performing pylorectomy a portion of the pancreas may be removed in making a wide dissection. I have had two such cases without any harmful result.

Young[2] reports 2 cases of injury to the pancreas during nephrectomy. He states this is more liable to happen on the right side, because the head of the pancreas is more fixed and approaches nearer the suprarenal body. In 1 case death occurred on the fourth day, and autopsy showed fat necrosis and acute pancreatitis. This was evidently due to the application of a clamp to secure bleeding at the bottom of the wound. Young says we should suspect such a condition where the operation is followed by rapid prostration and extreme distention.

Stuart,[3] in his review of cases of subcutaneous injury to the pancreas, cites 46 cases in which the results are known: 39 were operated; out of these 27 recovered and 12 died.

In several of the reported cases where operation has been performed the stomach has been described as being pressed forward and spread out over the cystic tumor. In each of these cases, had aspiration been resorted to, the needle would have passed completely through the stomach before entering the cyst. Aspiration should never be employed in tumors of the upper abdomen.

In conclusion, we would direct attention to the posterior incision as being the simplest and most direct method of reaching and draining cysts or abscesses of the pancreas. If the diagnosis is not made before exploration, then, as soon as the condition is clear, it is better to close the anterior incision and approach the tumor through an incision parallel to the lower border of the twelfth rib of the side on which the tumor is most prominent.

[1] Nebraska Med. Jour., January, 1920.
[2] Jour. Urol., 1917.
[3] Loc. cit.

FURTHER DATA CONCERNING THE EXPERIMENTAL PRODUCTION OF PANCREATITIS

By EDWARD ARCHIBALD, M.D.
MONTREAL, CANADA

ASSISTED BY

E. C. GIBBONS, Med., '23

The problem of the exact cause of pancreatitis and of the exact mode in which that cause works is still somewhat unclear. As you know, there are three principal theories. According to one, the pancreatic lesion is caused by the entrance of bile into the pancreatic duct by reason of an obstruction, either stone, or shred of mucus, or spasm of the common duct sphincter, at the outlet of the common duct; according to the second, which in this country is associated particularly with the name of Deaver, the lesion is caused by an infection traveling from the inflamed gall-bladder along lymphatics to the head of the pancreas; and the third theory is that duodenal contents are forced into the common duct through the papilla, temporarily relaxed, and so into the pancreatic duct. Of these theories the first and the third may be considered as proved for a considerable proportion of the cases reported, at any rate of the acute cases; while the second is still under discussion, and in any event is hardly susceptible of absolute demonstration. It lacks as yet experimental proof, depends upon purely clinical inferences, and goes, on the whole, contrary to the rules of pathology.

It is true that the entrance of duodenal contents has also never been demonstrated by animal experimentation. Nevertheless,

the conclusion that pancreatitis is sometimes brought about in this way imposes itself. I refer to cases in which postmortem dissection has shown that the pancreatic duct opened directly upon the mucous surface of the duodenum and not into the common duct, and to one or two rare cases in which there has been demonstrated localized pancreatitis of that part of the organ drained by the small duct of Santorini. These conditions, however, are fulfilled but rarely; and most of our evidence, both clinical and experimental, goes to show that the entrance of bile into the pancreatic duct is by all odds the most frequent mode of causation. This hypothesis has now got outside the bounds of mere theory and is widely accepted as being definitely proved.

I need not in this place recount the history of the clinical and experimental work which has been published in support of this proposition, but shall confine myself to recalling to your minds certain outstanding facts of the experimental work which has engaged my attention for the past few years, and which I presented before this Society two years ago. It was proved that in cats, in which animals the anatomical relationships of the common and the pancreatic ducts resemble those present in the human much more exactly than they do in other laboratory animals, it was possible to run a solution from the gall-bladder under a pressure of from 300 to 600 mm. of water into the pancreas to such an extent that even the finer radicles between the acini were found filled with the solution. In later experiments it was found possible in the same way to run bile, infected or not, solutions of bile salts, and mucin-free bile, from the gall-bladder into the pancreas with consequent production of various grades of pancreatitis from the least severe up to the acute hemorrhagic form, with death inside an hour. In all these cases the only obstruction possible lay in a resistance of the common duct sphincter.

From a consideration of these experimental facts and of clinical experience it seemed justifiable to present the problem of the etiology of pancreatitis in the following way: "The conditions for actual serious damage to be done in the pancreas must pretty certainly be three: (1) a change in bile composition, increasing the

proportion of bile salts; (2) undue resistance, perhaps often amounting to spasm, of the common duct sphincter; and (3) abnormal rise of pressure in the biliary system behind, either in the gall-bladder or in the common duct.[1]

"Our problem, therefore, is to discover in the facts of clinical experience circumstances fulfilling these postulates. Here we come on to uncertain ground. It is probable that hyperacidity may have a good deal to do with it. Symptoms indicating hyperacidity are frequently found in the previous history of patients with pancreatitis (Egdahl). Certainly the injection of hydrochloric acid into the duodenum or even into the stomach (Oddi) will cause a spasm of the sphincter. In man we are still unacquainted, so far as I know, with the condition of the duodenal contents as regards the length of time which is necessary for neutralizing the acid chyme of the stomach. In alcoholics, anyhow, and in patients who have duodenal ulcers, it would seem probable that the duodenal contents might remain acid for some time, and in these two classes pancreatitis is rather frequent. One recalls in this connection 3 cases published by Dr. William J. Mayo, in which acute pancreatitis was present, without gallstones anywhere, but with, in each instance, a duodenal ulcer situated close to the papilla, presumably causing hyperacidity.

"In the second place, that change in bile which results in a high concentration of bile salts and a diminution of bile mucin is probably brought about by the effect of gall-stones, especially when associated with inflammation. In infected bile, it is pretty certainly not the bacteria acting in their infecting capacity that cause the pancreatic lesion, but rather the chemical change in the bile produced by the action of bacterial growth. This at least was strongly suggested by the work of Flexner, of Carnot, Hlava, and others; and the present experiments, in which sterile bile and a solution of sodium taurocholate caused the lesion equally with infected bile, tend to confirm this view. I am unacquainted with any work upon the chemical composition of

[1] Archibald: Experimental Pancreatitis, etc., Surgery, Gynecology and Obstetrics, June, 1919.

infected bile, but it may be presumed that the action of bacteria is to precipitate the mucin of bile; and indeed we see the evidence of it in the shreds observed in the bile in cases of cholecystitis and cholangitis. If this is the case, it is probable, contrary to the assumption of Flexner, that the relative proportion of the bile salts is increased. I may add that Dr. Harding, associate professor of chemistry in McGill University, is beginning work along this line. It must be pointed out, however, that the effect of bacteria upon the bile seems to increase very greatly the destructive effect of that bile upon the pancreas; and while this is still probably due to some chemical change in the bile, it seems likely that there come into play, in addition to the bile salts, new substances possessing necrosing properties.

"It may be noted also that any obstruction in the cystic duct would likely deprive the bile of a part of its mucin content normally provided by the gall-bladder mucosa; and in such cases, theoretically at least, the occurrence of a pancreatitis may be rendered more likely.

"In the third place the condition of increased pressure in the biliary system is presumably brought about partly by an increased resistance of the sphincter, set in motion by hyperacidity or by neighboring ulcers; partly by a sudden blocking of the cystic duct by stone or inflammation; and finally, perhaps, by any unusual increase in the amount of bile secreted by the liver. The effect of a full meal, two or three hours after which, as we see in so many case reports, the attack of pancreatitis is apt to come on, may be in the direction of increased bile production rather than in that of increased pancreatic secretion."

The present work represents a small effort to get deeper into these problems. Fourteen cats have been used. A later report will give details of the experiments performed. Briefly, we have injected ox bile, infected with various organisms, as well as human bile, aspirated at operation from acutely inflamed gall-bladders, into the gall-bladder of cats, under pressure, either by running it in from a raised funnel or by squeezing of the gall-bladder. In most, the bile entered the pancreas. We have also injected by

syringe direct into the pancreatic substance cat's own bile, human bile from distended inflamed gall-bladders, in one case complicated by very acute pancreatitis, the same human bile several days later running clear and normal-looking from the cholecystostomy tube. We have reopened at intervals, cut out sections of pancreas for microscopical examination, and have taken cultures from the interior of the pancreas, often recovering the original organism, thus proving penetration of bile. We have injected HCl into the duodenum to stimulate the sphincter.

Microscopically we have found all grades of pancreatic necrosis of the parenchyma, with much or little stroma reaction, edema, congestion, hemorrhage, going on after from one to four weeks to advanced replacement fibrosis, local or diffuse. We could follow the development of interstitial pancreatitis, a pancreatic cirrhosis from the original necrosis, through fibroplastic repair.

The investigation has been directed chiefly along the following lines. In the first place, what effect does infection in bile produce upon the relative proportions of the bile salts and the mucin? Secondly, what difference, if any, exists between the action of infected bile on the pancreatic parenchyma and that of normal bile? Third, is it necessary that an abnormal degree of the force which expels the bile should be present before the pancreatic duct is invaded, and if so, what is the nature of that abnormal force? Finally, under what condition is the common duct sphincter brought into resistance to the flow of bile into the duodenum?

I. The Chemical Examination of Infected Bile. During the past winter Mr. Logan, working in the chemistry department of McGill University, under Dr. Harding and Professor MacCallum, has been analyzing, according to the methods of Foster and Hooper, samples of bile which I have brought him from the wards of the Royal Victoria Hospital, together with specimens of bile from the ox, the hog, and the cat. These biles have been variously infected. My idea was that as the result of infection the mucin of bile might be precipitated in such an amount as to leave the bile salts in a much higher proportion, and greater concentration, than in normal bile, and that such a

bile being forced into the pancreas would presumably cause a more definite and more severe pancreatitis. Flexner showed that it was the bile salts that caused pancreatic necrosis, while the mucin had a protective action. Our work leaves this supposition as yet unconfirmed. Clinical experience, on the whole, seems to indicate that the more serious cases of pancreatitis are associated with conditions of serious inflammation in the gall passages. Mr. Logan's work is still very incomplete. The difficulties of the subject are much greater than the uninitiated might suppose, and I am unable to make any very definite report upon this part of the work. On the one hand the actual amount of bile salts excreted in the bile varies very greatly according to the nature of the diet, chiefly, but varies anyhow on a mixed diet from day to day (Hooper and Foster). On the other hand the amount of mucin in a normal fistula-bile remains fairly constant. The amount of mucin, when the bile has been infected, shows in some cases a slight increase, in others a slight decrease. The analyses, so far, which have been carried out with several of the ordinary organisms, staphylo- and streptococci and B. coli, are insufficient in number to give any sure indication on this point. We expect to carry on the work during the coming year. But for the present it looks as if our hypothesis as to the greater concentration of bile salts as the result of infection remains unsubstantiated. There is probably something else in infected bile which is responsible for the more severe necrosing effect which it shows as compared with normal bile. Upon this point several of the experiments are most positive. One of these in detail is as follows:

Cat 190. Bile aspirated from gall-bladder at operation on a case of acute cholecystitis with impacted stone in cystic duct. The bile was blackish green and was obviously infected. Culture showed a scant growth of streptococcus. The gall-bladder was drained. A portion of the original infected bile was injected with a syringe into the splenic end of a cat's pancreas, and another portion of the drainage bile, which three days after operation had become clear and normal looking, was injected into the duodenal end of the same pancreas. After two days the cat was

reopened, and it was found that the infected bile had caused a very marked inflammatory reaction, with fat necroses in the adjoining omentum. In marked contrast with this, the more normal drainage bile had left the duodenal end almost normal. These two areas were cut out and sectioned, and the difference in necrosing effect was confirmed. At the splenic end the necrosis of parenchyma was extensive and there was a great deal of edema and congestion, while at the duodenal end there was very slight necrosis, little edema, and practically no congestion.

This observation was confirmed in two other cases of acutely infected bile from cases of acute cholecystitis, and in still another one the same contrast was found between the effects of cholecystitic bile and the cat's own normal bile.

The difference is one of degree. Even normal bile entering the pancreas causes necrosis of the pancreatic parenchyma with which it may happen to come in contact. But this necrosis is aseptic, is accompanied by little, if any, surrounding reaction, whereas the infected bile causes a more massive and extensive necrosis with a great deal of edema, congestion, even hemorrhage, and with much inflammatory response on the part of the fixed connective tissue. Fat necroses also are often more abundant.

While these results were obtained by the direct injection of bile through a needle, we were able to determine that similar results, and like differences, were found when the bile entered through the pancreatic duct from the gall-bladder. It may here be said that in the present series, as in that of two years ago, in a majority of the experiments in which any pressure was applied from behind, that is on the gall-bladder, the bile entered the pancreatic duct to some extent, as was evidenced by the finding of necrosis. In some instances the effect seemed localized to the central part of the gland nearest the entrance of the pancreatic into the common duct, while in others the bile had invaded the whole gland.

The degree of swelling of the organ was variable. One fact in particular is noteworthy, which was that there might be macroscopically very little evidence of any lesion in the pancreas as

to swelling or congestion, and yet microscopically necrosis might be quite extensive. In all such cases the bile was practically normal. In other cases, and these the majority, the effect of bile entrance was evident in a definite edema and swelling, with slight induration of the organ. In these, on microscopical examination, one may find all grades of necrosis, diffuse or localized, involving a few acini or whole lobules, the individual cells showing early stages of degeneration—a diffuse "cooked" appearance—or advanced stages of mass necrosis together with marked edema and congestion. These observations are, I think, important from the clinical standpoint. We are all of us frequently troubled at operation to decide whether the pancreas, which upon palpation we think is somewhat enlarged, a little harder than normal, and nodular, is really the seat of a mild inflammation or is normal. The pathologists tell us that in such cases, upon the opportunity of a postmortem, they can find no evidence of pancreatitis, and clinicians of prominence warn us that we should be very chary of saying that there is actual pancreatitis present. In two patients, suffering from quite definite subacute pancreatitis, one of my own and one of Dr. Garrow's, a small section had been taken from the pancreas at operation for microscopical examination. Let me repeat that the pancreas to palpation was quite certainly enlarged and hard. In both the pathologist was of the opinion that there was no clear and definite pathological change present, yet in both I felt justified in concluding that there was clear evidence of pancreatic damage. In certain areas the cells were disintegrated, cell membranes broken, nuclei swollen, cytoplasm poorly staining, and in one or two places actually necrotic. There was also marked edema and some congestion. I felt sure that these were not artefacts or due to imperfect technic, as the pathologist was inclined to believe, because I had seen the same slight lesions in association with absolutely definite lesions so many times in animals. I would, therefore, suggest that at least in many of the cases in which, at operation, we feel that the pancreas is *probably* swollen, it really *is* swollen; and that this effect is due to the entrance of small quantities of

bile not seriously infected, and constitutes a warning that the biliary system must be freed of all possible infection.

The second part of the problem concerns the question of the *vis a tergo*. It is probable that before pancreatitis of any severity can be caused, there must be an increase of pressure in the expelling forces of bile. A number of investigators (Doyon, Freese, Mann, Bainbridge and Dale) have investigated the contractile force of the gall-bladder in animals and give figures approximating 150 to 225 mm. of water. An exact estimation is difficult, and they were concerned only with the normal animal. It is probable that in the human, and under pathological conditions, the contracting force of the gall-bladder may be much increased. Dr. F. N. G. Starr reports the following very remarkable observation: During operation on a patient suffering from cholecystitis with stones, under very light anesthesia, he inserted his finger into the neck of the gall-bladder and immediately found it grasped apparently by a very strong muscular contraction, so strong that, as he puts it, he would have pulled the whole liver out if he had exerted his strength to pull his finger out. It was apparently a severe reflex spasm in the semiconscious patient. It may be that, if we were not accustomed to operating under full ether anesthesia, we might encounter the same experience upon occasion. In any case this observation seems to me to have a direct bearing on the question. If, as the result of the stimulus of stone and inflammation, the gall-bladder is able to contract violently, we are immediately provided with the driving force needed to push bile into the pancreas. In my experiments it has been, as a rule, necessary to fill the gall-bladder more or less rapidly under pressure, or to give it a few squeezes, in order to stimulate the sphincter to contract and offer the requisite resistance. The normal contraction of the gall-bladder is undoubtedly feeble, and, according to Meltzer's law, the sphincter presumably relaxes at the same moment, so that ordinarily bile is not forced into the pancreas.

Another observation, which I owe to Dr. Mann, is that upon any sudden descent of the diaphragm with fixation of the abdom-

inal walls, as in sneezing or vomiting, the pressure in the gall-bladder is driven up to a great height. This also might have some bearing on the question.

In three animals I have tried the effect of eserin hypodermically, and of barium chloride painted on the surface of the gall-bladder. In no case did I observe any peristaltic movement in the gall-bladder wall, although with a very high dose of eserin there seemed to be a slight tonic contraction of the gall-bladder as a whole.

With regard to the third factor, the abnormal resistance of the common duct sphincter, I have nothing new to add. One thing seems definite, that acid in the first and second portions of the duodenum will cause a temporary spasm of the sphincter. The contents of the duodenum have in the last few years been frequently examined since the use of Einhorn's tube has become general, and I understand from Dr. Einhorn (verbal communication) that sometimes he has found the duodenal contents to remain acid for a certain time. Whether one should, in cases of duodenal ulcer, or of the gastroduodenitis of the alcoholic, expect such a condition to be frequently present I do not know, but at least such an assumption is not unlikely, and in that case one might expect a recurring spasm of the sphincter; and this might serve to explain the frequency of pancreatitis in association with ulcer and alcoholism. This must still remain a matter of speculation. In any case, so far as my experiments go, it would seem that the more important factor in stimulating the sphincter to resistance lies in a sudden rise of pressure from the gall-bladder side, rather than in irritation from the duodenal side; and in this connection I am inclined to believe that the reason why a cholecystectomy is apt to cure coincident pancreatitis, lies partly in the fact that the only serious muscular contractile force in the biliary system has been cut out, and partly, also, in the fact that with the removal of the gall-bladder there is also removed the source of infected bile, at least in most cases.

SUMMARY. 1. Infected bile, aspirated from the inflamed gall-bladder, exercises a much more severe necrosing and inflammatory effect upon the pancreas than does normal bile.

2. Chemical investigation of infected bile has not yet proved that this difference of effect is due to an increase in concentration of the bile salts as the result of bacterial action on the bile.

3. Mild grades of pancreatic swelling, as estimated clinically, are certainly possible, and are represented by edema with early necrosis of the parenchyma, presumably the result of bile invasion. The clinical statement in operation records as to the presence of "a somewhat thickened and indurated pancreas" is probably a correct interpretation of fact in most instances.

4. The gall-bladder, under conditions of irritation from stone or inflammation, is probably able to go into strong muscular contraction; and the hypothesis is set up that such contractions may provide sufficient driving force to cause invasion of the pancreas with bile.

5. The common duct sphincter is provoked to resistance not only by an acidity of the duodenal contents, but also by a sudden distention of the common duct through abnormal and unexpected rises of pressure in the gall-bladder.

THE SURGICAL REMOVAL OF PANCREATIC STONES: A REPORT OF CASES

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PANCREATIC stones occur so infrequently that one is seldom called upon to remove them surgically. A review of the literature shows very few cases in which a pancreolithotomy has been performed. It is probable that such stones are often overlooked during the course of abdominal explorations, as they have been found at autopsy on a number of occasions. The consistency of the pancreas is probably responsible for the difficulty with which they are recognized at operation. When multiple stones are present a sense of crepitation is brought out upon palpating the gland, which makes the diagnosis easy; but single stones are difficult to recognize when palpated through the pancreatic tissue. In reviewing the histories of the patients operated upon for pancreatic calculi in the Mayo Clinic, I find that during eleven years this operation has been performed only four times. It is customary in this clinic to routinely examine the pancreas in all patients operated upon for upper abdominal disease, and the fact that so few pancreatic calculi have been found by surgeons who routinely examine the pancreas during this time shows that pancreatic calculi are either extremely rare or very difficult to recognize at operation. The presence of stones in the pancreas was first reported by Graaf in 1667, and the first report of the surgical removal of stones was probably that of Mr. Pierce Gould in 1896. Since then case reports have occasionally been published, but to the present time the list of reported cases is extremely small.

Pancreatic calculi very closely resemble the calculi found in the ducts of the salivary glands. They are usually of a white or slightly gray color, fairly firm in consistency, brittle and irregular in shape; however, they may be facetted on a portion of their surface, as was seen in the stones removed from one of our patients. Chemical analyses which have been made have shown that they are composed of carbon salts, phosphorus or magnesium salts and organic matter.

As there is still considerable controversy as to the etiology of pancreatic diseases it is impossible to say just how these stones are formed and what part they might play as a factor in pancreatic disease. It would seem, however, that the manner of their formation must be very similar to that of stones in the biliary tract, that is, from infection in the ducts or from the stagnation of pancreatic fluids from obstruction.

Diabetes has often been observed associated with pancreatic stone. Hansemann in 72 cases of diabetes associated with pancreatic disturbances found stones in 12 cases, and Oser in 70 cases of lithiasis observed that diabetes was recorded 24 times. It is difficult to say just what part, if any, these stones played in the production of the diabetes. It seems most likely that both the diabetes and the stones had resulted from the chronic inflammatory changes in the pancreas, but it is possible that the diabetes may have resulted from the chronic inflammatory changes in the gland produced by the calculi.

Pancreatic calculi have been reported in some instances where pancreatic cysts were present and it is probable that they are responsible for the formation of certain of these cysts.

The symptoms produced by stones in the pancreas are so very similar to those seen in chronic pancreatitis associated with stones in the biliary tract that a definite diagnosis of their presence in the pancreatic ducts is impossible. The presence of stone may be suspected and several cases have been reported in which such a diagnosis was made and was later confirmed at operation, but a definite diagnosis can be made only at operation. On account of this difficulty in diagnosis, the pancreas, during

the course of operations for infection or stones in the biliary tract, should be carefully palpated for evidences of stone.

Pancreatic calculi may be single or multiple or may entirely fill both of the pancreatic ducts. They also have been found near the periphery of the gland, where they have formed in the small ducts. When they are located in the ducts near the point where these empty into the duodenum they may often be recognized at operation by careful palpation; when multiple stones are present they may be recognized by the peculiar feeling of crepitation which they impart to the palpating hand upon manipulation of the gland. It is difficult to decide definitely as to the character of localized tumors in the head of the pancreas, as these may be due either to neoplasms or to impacted stones surrounded by an area of inflammation. In one of the cases operated on here, as in a case reported by Link, a distinct ridge was present on the anterior surface of the gland which corresponded to the course of Wirsung's duct, which could be felt throughout the entire length of the pancreas.

Up to the present time very few operations have been performed upon the pancreas proper, and in most instances the chronic inflammatory diseases of this gland have been treated indirectly by drainage of the gall-bladder or common duct. Experimental work done by Opie, Coffey, Sweet and others tends to show that surgery of the pancreas is not necessarily attended with the risk commonly supposed to be present. Operations for the removal of pancreatic stones present no unusual difficulties except in instances where the stones are impacted in the ducts near the ampulla, and, I believe, can usually be performed safely. When operating for stones the pancreas should be exposed by the route which seems to offer the best exposure of the gland. If the stomach is high, exposure can be best obtained through the gastrocolic omentum; while if the stomach is low, it can best be exposed through an opening in the gastrohepatic omentum. The pancreas also has been exposed by lifting up the omentum and colon and making an incision through the transverse mesocolon. Stones have been

removed from the lower portion of the duct of Wirsung through an incision through the mesentery of the duodenum, and cases have been reported in which the anterior wall of the duodenum has been opened and stones removed by incising the papilla

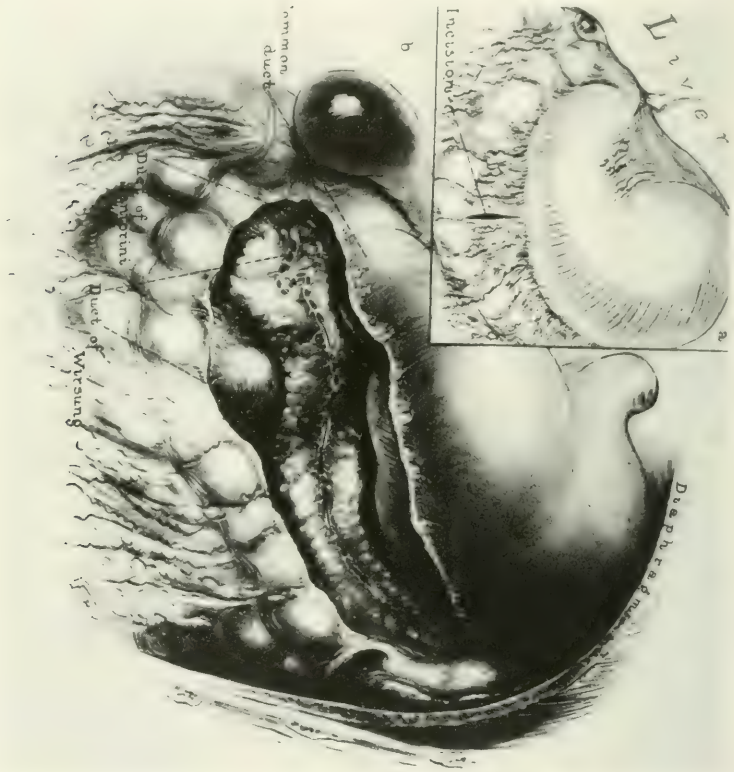

FIG. 1.—*a*. Incision in the gastrocolic omentum. *b*. Opening in the lesser peritoneal cavity and incisions along the course of the pancreatic duct. The gastrocolic omentum has been divided extensively in order to show the entire pancreas.

of Vater and the posterior wall of the duodenum, thus exposing the ampulla and the end of the pancreatic duct. Following the removal of calculi, hemorrhage may be easily controlled and the opening in the duct closed without difficulty with small silk

sutures. In the case reported by Link the tail of the pancreas was drawn out through an opening made in the transverse mesocolon, and after the stones had been removed a catheter was used to drain the pancreas, the tail of which was drawn out through the abdominal incision.

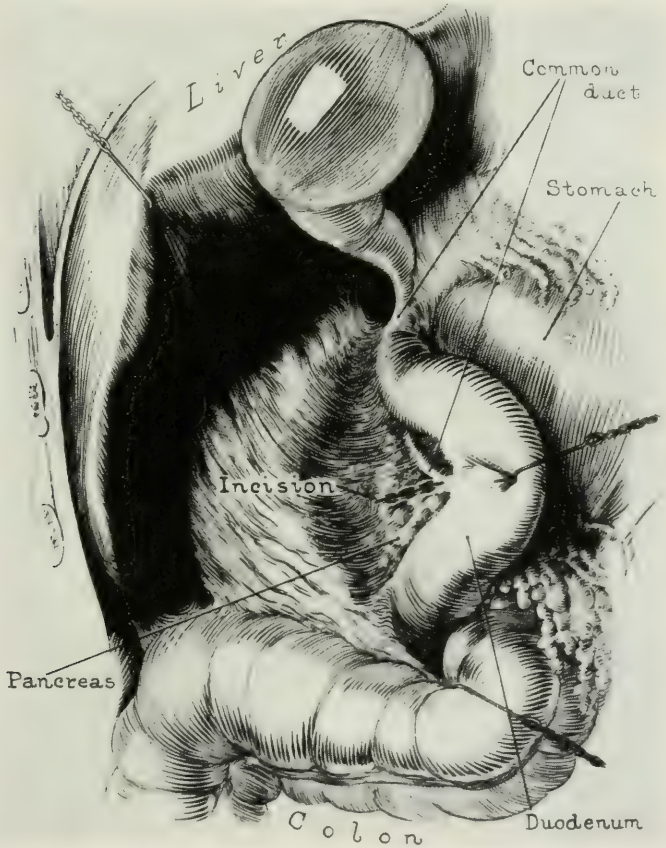

FIG. 2.—The mesentery of the duodenum is incised and the duodenum turned back in order to expose the head of the pancreas.

The ducts of the pancreas are without valves, and in cases of obstruction where pancreatic drainage is thought advisable it is

possible that an operation of the type described by Link or that described by Coffey, in which the cut-off end of the pancreas is

FIG. 3.—(Case A288735). Pancreatic stones.

transplanted into the jejunum, might be done. In the four cases operated on in the Mayo Clinic the openings in the pancreatic duct were closed and rubber tissue drains used to take

care of any pancreatic fluids which might escape. The experimental work of Opie, which has been confirmed by Coffey and others, has shown that fat necrosis occurs only when pancreatic fluid is poured into and confined in the retroperitoneal fat; when pancreatic fluid is delivered inside the unbroken peritoneum, fat necrosis does not occur, and no harm can result from this provided sufficient drainage is placed to readily conduct the pancreatic fluid to the surface. It would seem safest, therefore, in operations where the pancreatic ducts are opened, that some provision be made for carrying off the pancreatic fluid in case leakage should occur.

I wish to report in this paper four cases operated on in the Mayo Clinic since 1910:

Case I.—Female, aged forty-eight years. For fourteen years the patient had attacks of sudden, sharp, cramp-like pains in the epigastrium, radiating through to the back, accompanied by nausea and often by deep jaundice; for many years a good deal of flatus and diarrhea, often having as many as twenty-five stools, without blood or mucus, in twenty-four hours. Jaundice for one year. Considerable weight and strength loss. A diagnosis of gall-bladder disease with probable common duct stones was made. Operation June 16, 1910. A stone the size of a filbert and several smaller ones removed through an incision in the anterior wall of the duodenum; this stone found in a diverticulum in the head of the pancreas just off from the common duct at a point about one-half inch above the papilla. Ampulla opened for distance of about one-half to three-fourths of an inch. Gall-bladder largely destroyed; remnants of it removed. Spleen four times normal size. Patient also had pyloric obstruction from chronic duodenal ulcer and at another operation a gastro-enterostomy was made. Two years later she was in excellent health.

Case II.—Male, aged thirty-three years. History typical of gall-stones for several years. One year previously operated

upon elsewhere with removal of stones from gall-bladder and common duct; drainage of gall-bladder. Pancreas found to be enlarged and thought to contain stones, but no attempt to remove them. When examined in Mayo Clinic patient found to have diabetes; sugar disappeared from urine under treatment and strict diet. Operation October 1, 1919. Enlarged gall-bladder which was free from stones. Two stones removed from common duct. Pancreas slightly enlarged and quite hard. Peculiar crepitating feel imparted to hand upon palpating pancreas, and a ridge could be felt on anterior surface of the gland corresponding to the course of the duct of Wirsung, which extended from the tail to the head. Pancreas exposed through opening in gastrocolic omentum and many stones removed through four incisions in the duct of Wirsung. Several stones in the duct near the ampulla removed through an opening made through mesentery of duodenum. Openings in the duct closed with small silk sutures. Gall-bladder and common duct drained. Several rubber tissue drains. Duct of Santorini seemed filled with stones, but on account of extent of operation already performed no effort made to remove these stones. Diabetic condition improved considerably after operation and sugar tolerance became much increased. Lately patient has had symptoms indicating that infection is still present in biliary tract.

Case III.—Female, aged sixty-eight years. Attacks of epigastric pain radiating to left side but not to the back for some years. Fifteen pounds weight loss within past year. Appendix found to be definitely diseased. Multiple stones could be palpated in head of pancreas. These removed through opening made through mesentery of duodenum. Stones appeared to be in main duct of pancreas but were near surface. Uneventful recovery. No subsequent notes.

Case IV.—Female, aged sixty-two years. History of ulcer of duodenum for fifteen years. *X*-ray showed pyloric obstruction. Unable to obtain history of gall-bladder trouble. Operation

January 1, 1921. Chronic perforating duodenal ulcer with pyloric obstruction found. A stone could be felt by palpation in pancreatic duct near ampulla. Opening made in duct through mesentery of duodenum and two stones size of navy beans removed. Gastroenterostomy made. Good recovery. No subsequent notes.

DRAINAGE OF ABSCESS OF PANCREAS

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The object of this paper is to direct attention to a very simple and safe method of draining an abscess located in the tail of the pancreas. The history of the case in the management of which this form of treatment suggested itself is not of sufficient interest to warrant its publication in full.

A woman, aged forty-eight years, with symptoms of ulcer of the greater curvature of the stomach, which later proved to be malignant, was subjected to an exploratory laparotomy which demonstrated an indurated mass 5 cm. in diameter in the posterior wall of the stomach attached to the tail of the pancreas, which was swollen to the size of an orange, about 10 cm. in diameter. This swelling was congested and contained fluid. Evidently an abscess had formed in the tail of the pancreas from an infection originating in the perforated gastric ulcer.

In order to determine the condition more perfectly, an opening was torn in the transverse mesocolon and the abscess was found strongly adherent to the posterior wall of the stomach while the posterior wall of the pancreas was quite free. Great care was exercised in the manipulation of the pancreas not to rupture the abscess. An incision was then made in the left flank, immediately below the last rib, 5 cm. long, and a pair of forceps passed forward, guided by one hand, in the abdominal cavity to a point behind the pancreas. The blades of the forceps were spread widely open in order to establish a broad passage. The space behind the pancreas was then loosely packed with gauze, in the

middle of which was placed a large rubber drainage tube. The gauze and the drainage tube were passed out of the wound in the flank and two cigarette drains were added and carefully placed behind the pancreas. The tear in the mesocolon was then repaired and the abdominal wound was closed. Five days after the operation the abscess broke spontaneously and a large amount of thick pus escaped. The gauze and the cigarette drains were removed gradually. The rubber tube was left in place for two weeks, when the discharge had been greatly reduced. The tube was then removed and the wound healed in two more weeks.

In case the abscess had not opened spontaneously it could have been opened safely after adhesions had been formed around the gauze tampon by passing long dressing forceps through the large drainage tube into the abscess, when the tube could have been pushed forward into the cavity of the abscess. The method is so simple and safe that it seems worth bearing in mind.

PREPANCREATIC AND PANCREATIC DISEASE

By JOHN B. DEAVER, M.D.
AND
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(By Invitation.)

The lower abdomen has yielded its most obvious secrets to the persistence of the internist, the pathologist and the surgeon, but the upper abdomen still remains in many respects a terra incognita that not only fascinates the explorer but calls urgently for elucidation of important questions in the interest of our patients. Not the least of these problems are those associated with pancreatitis. The treatment of this condition is on a very insecure footing at the present time and exact knowledge of the etiology and pathogenesis would help materially in crystallizing our ideas as to the proper procedures. In the meantime we must act according to our lights and be alert with clinical observation, experiment and pathological study to clear up the subject. The difficulty in coming to a conclusion concerning the cause of pancreatitis is due to the multiplicity of factors involved. In addition to infection and duct obstruction it is necessary to consider in connection with pancreatic disease the peculiar role of the ferments elaborated by the gland itself, the conditions under which they may be liberated and activated within the tissues and the anatomic variations which may contribute to this result.

The earlier observations emphasized the factor of obstruction, beginning with Opie's classical observation of acute pancreatitis associated with impaction of a gall-stone in the papilla of Vater. This case led him to advance the hypothesis that bile, being thus

prevented from passing out into the duodenum, might be driven up the pancreatic duct provided that the latter opened into the common duct or the papilla of Vater proximal to the stone. Opie showed further that in a percentage of cases the anatomy of the bile and pancreatic ducts was actually so constructed as theoretically to permit of this occurrence. Actually the entrance of the pancreatic duct—as with other ducts of discharge throughout the body—is oblique, and guarded not only by this valvular formation but by a sphincter in such a manner that doubt is cast on the probability of such retrojection under clinical circumstances. Still, in the presence of anatomical abnormality or inflammatory disturbance of the duct opening, the possibility must be admitted. Moreover, Opie showed that injection of the bile under pressure into the pancreatic duct experimentally did produce acute hemorrhagic pancreatitis, which was promptly verified by Flexner and many others. It was shown, too, that infected bile was more potent than sterile bile in producing the lesion. This seemed to be a complete and satisfactory chain of proof of the etiology and pathogenesis of acute pancreatitis and was suggested also as a probable cause of chronic pancreatitis, the result of less violent and more frequently repeated insults to the pancreas by way of its ducts.

Gradually, however, it became clear that this was not the whole story. Very few cases of acute pancreatitis showed a stone impacted in the papilla of Vater. Many cases (varying from 20 per cent to 50 per cent in various series) showed no stone at all in any part of the biliary tract. Naturally it was impossible for the surgeon under clinical conditions to investigate the anatomic relations of the ducts, and pathologists in general have neglected their opportunity in cases coming to autopsy (a point which should be followed with the greatest keenness in the future). But at least one instance, that of Bobitzki, is on record where acute pancreatitis was found in a case in which the ducts did not join but emerged side by side into the duodenum. There was no possibility of retrojection of bile in this case, unless it was directly from the duodenum with other duodenal contents. This also

was mentioned as a possibility, notably by Hlava, and Seidel claimed to have demonstrated it experimentally. Other observers, however, have been unable to verify his results, the opening of the duct into the duodenum being competent to resist a force sufficient to rupture the duodenum. In fact, the greater the distending force, the more secure was the closure of the duct. Here again anatomical or pathological anomaly may permit this mechanism to operate. Beall has recently reported and pictured a case in which *x*-ray examination following the administration of bismuth by mouth seemed to show this material in the common and hepatic ducts. However, the evidence in reality argues against such a method of causation, since such examinations are nowadays being made in vast numbers without evidence of regurgitation of duodenal contents into the bile or pancreatic ducts. The frequency of pancreatic disease, therefore, does not parallel the very unusual character of this isolated observation. Extension of experimental work has shown that the greatest variety of substances injected under pressure into the duct could set in motion the pathological processes characteristic of acute pancreatitis. Moreover, direct massive traumatism and interference with blood supply, if sufficiently extensive, furnishes a very suggestive parallel to certain clinical conditions. The underlying efficient cause of all such experimental conditions was well summarized by Sweet as residing in damage to the pancreas resulting in interstitial liberation of its ferments, plus activation of those ferments, which might occur *in situ* by bacterial activity, autolysis of tissues or by the action of various substances, bile or duodenal secretion being no more efficient than others.

A priori, also, it seemed strange to adopt an explanation of all the inflammatory diseases of the pancreas which, for this organ alone among the organs of the body, eliminated entirely or almost entirely the influence of infection. Now, during the period of development of the subject by Robson, Mayo, Moynihan and others, surgeons came to recognize the great frequency of the association of inflammatory lesions of the important organs of the upper abdomen, and to note that the pancreas frequently

participated, as shown principally by enlargement and changes in consistency of the head of the gland, the remainder being often unaffected so far as could be determined under the difficulties of thorough examination. Bartels and Franke had previously shown by anatomic investigation the extraordinarily close relationship which exists between the lymphatics of the gall-bladder and the adjacent duodenum. Arnsperger suggested its clinical application, and Deaver and Pfeiffer, at operation, have noted and in repeated communications called attention to Nature's demonstration by pathologic injection of the train of events, namely, infected gall-bladder, infection of the lymphatics coursing down the gastrohepatic omentum often presenting greatly enlarged glands in this location, and finally glandular and lymphatic involvement about and directly on the head of the pancreas. No tissue so surrounded by an inflammatory process could escape participation at all times, and the wonder seems to be that it does escape so often and in so many instances is able to return to normal when the existing cause is removed. For this the well-known relative immunity of the pancreas to infection must be given credit, as showing the ability of the pancreas to deal with inflammatory processes. An early observation of Kehr is significant. Just before the significance of fat necrosis was explained, he operated upon a case which showed this phenomenon but neglected to examine the pancreas. A year later he had occasion again to open the abdomen and found the pancreas normal in appearance and to palpation.

Recently, Rous and McMaster, in an interesting piece of work which seems to throw much light on the function of the gall-bladder, showed the great activity of the organ in abstracting from the bile its fluid constituents, thus explaining the common finding of inspissation of the bile in the gall-bladder as compared with that of the common duct. Still more recently, in an unpublished piece of work from Sweet's laboratory, the lymphatics are, as might be suspected, found to be active agents in absorption from the gall-bladder. When the bile and gall-bladder walls are infected it would seem that a torrent of infection is thus swept down into the retroperitoneal and peripancreatic areas.

Another point to which we have given consideration, but have not hitherto directed attention, is the fact that gall-bladder disease is commonly accompanied by choledochitis. Such inflammation is unquestionably propagated to the adjacent pancreas, where the duct pierces it in its terminal portion, which it does in two-thirds of all cases. This is also the portion of the pancreas through which the duct of Wirsung, its main avenue of discharge, passes on its way to the ampulla of Vater or the duodenum, as the case may be. Now, swollen pancreatic tissue exercises firm compression not only on the common duct, as is known, but also on its own duct, thereby causing, without need for other explanation, a certain amount of obstruction which would favor both ascending infection and probably, by increased pressure, the rupture of the minute canaliculi which convey the secretion from the pancreatic cells to the terminal ducts. It is probable that the rupture of these canaliculi is the factor common to many methods of producing pancreatitis experimentally.

Archibald has recently revived Opie's original explanation in new form. For the minute obstructing calculi impacted in the papilla of Vater, which it is now known is at least not always present, he has substituted the action of the sphincter of Oddi, which normally encircles the termination of the common duct. Given sufficient resistance by spastic closure of this structure, retrojection of the bile into the communicating pancreatic duct might occur, just as in blockage by stone. Archibald has detailed one experimental case in which he produced the picture of acute hemorrhagic pancreatitis in the cat by intraductal pressure applied through the gall-bladder, the only obstructing force being the normal muscle of Oddi. To us it seems fair to say, without going into too tedious detail, that his remaining protocols seem to have little bearing upon the problem, as they are unconvincing and admit of other explanations.

While it is necessary to be cautious, since pancreatitis has been produced experimentally in so many ways which do not operate clinically, and one case is a doubtful basis for claim, Archibald's work is of great interest in presenting another possibility by

which the underlying factors which produce pancreatitis may be set in motion. There is little disposition in any quarter to deny the probability of Opie's explanation in certain cases, and it is possible that Archibald may have added another mechanism of production. Oddi originally, and Mann, McWhorter and others more recently, seem to have demonstrated that Oddi's sphincter does influence the intraductal pressure of the bile. It would seem, if Archibald's observations were of great importance, that pancreatitis should be more frequent after cholecystectomy, which tends, temporarily at least, to heighten the pressure in the ducts, as shown by the frequent observation of dilatation after the operation. We have encountered no such case in the literature, unless the process was so recent as to arouse the suspicion that it was already present and undetected at the time the gall-bladder was removed. It may be claimed that the gall-bladder is the propulsive force necessary in forcible retrojection, but no observations, clinical or experimental, exist to show that the gall-bladder exercises other than a tonic influence on the pressure within the ducts.

Such is a brief summary of our present knowledge and the reasoned probabilities of the etiology and pathogenesis of pancreatitis. It will be noted that even the confirmed advocates of retrojected bile due to some form of obstruction as the sole and efficient cause of pancreatitis, admit and even stress the great importance of infection in the bile as a contributing cause of the condition. Those who start with infection as its ultimate cause do not deny the efficiency of a mechanical cause superadded, the mechanical causes themselves probably being dependent in the last analysis upon present or previous infection.

We must not, therefore, be diverted by mechanical explanations from our idea of proper treatment, which is to get rid of the infective process which is at the bottom of the whole train of events. Division of the sphincter of Oddi, which has been suggested by Archibald but never tried clinically so far as I know, seems to us to be attacking the problem at the wrong end. If organic stricture could be proved the situation would be different,

but the difficulties in the way of such demonstrations are great and subject to clinical dangers. We must continue to insist upon infection as the underlying cause of pancreatitis, whether acute or chronic, and to urge preventive, which is another word for early surgery.

One fact seems to have become sufficiently clear from clinical observation, namely, the need for prolonged drainage of the biliary tract in cases of chronic pancreatitis. The drainage, moreover, should be external and not internal. Theoretically it would seem ideal to anastomose the gall-bladder to the stomach or the duodenum, preferably the latter, in order to establish continuous drainage, which might save the double purpose of diverting the bile from the common duct and the ampulla and also enable the biliary tract the more readily to throw off any chronic infective process. If it worked out in this way it would answer the requirements of the advocates of both the obstructive and the infective methods of causation of pancreatitis. Unfortunately clinical experience shows that too often this happy result does not follow. In the absence of obstruction of the common duct there is a marked tendency for rapid closure and obliteration of the anastomosis, leaving the anatomy and physiology the same as before, except for the disadvantage of crippling adhesions which have been added by the operation. Our best results have been obtained by prolonged external drainage by way of the gall-bladder or the common duct. If the gall-bladder is delicate and relatively unaffected to gross examination, it is our practice to drain by this route, marsupializing the gall-bladder to insure longer drainage. Three months is the minimum period of drainage for this type of operation. If the condition of the gall-bladder is such as to indicate cholecystectomy, which is true of all cases of obvious interstitial disease, or if the symptoms have indicated intermittent obstruction of the common duct, or, finally, if at operation the common duct be found dilated, whether it contains stones or not, it is best to drain the common duct itself by means of a T-tube, which may be left *in situ* without fear of displacement as long as is considered desirable. In

these cases drainage should be maintained for at least three months—and better six months, or in some cases even longer. Such patients wear a small bottle supported by a belt or girdle. The end of the tube is placed in the bottle and the small amount of drainage after the patient becomes ambulatory occasions no inconvenience. Before deciding to remove the tube it is wise to clamp or ligate it for a week or more. It can then be seen whether the patient still needs this safety valve. It is much easier and safer to leave it in than to put it back, by operative means, if it transpires that the drainage period has not been long enough. We have observed no ill effect from the prolonged drainage—our regret having been, in more than one instance, that the tube had not been allowed to remain sufficiently long.

The success of this plan of treatment confirms us in our belief in the infective origin of this disorder, since drainage certainly can cure infection but it cannot change the anatomy of the terminal ducts.

Finally, chronic pancreatitis is to be regarded as ordinarily a complication of preëxisting surgical disease, and in that sense is a reproach to our diagnosis and therapeutics. If certain modern gastroenterologists are to be allowed to have their way we must expect to have more of it in the future.

OBSERVATIONS ON THE PATHOGENESIS OF INFECTIONS OF THE BILIARY TRACT[1]

By EVARTS A. GRAHAM, M.D.

AND

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(By Invitation.

Present conceptions of the pathogenesis of infections of the gall-bladder and bile tracts are based on four assumed possibilities: (1) Descending infection from the liver by bacteria carried down in the bile; (2) ascending infections from the duodenum up the lumen of the common bile duct; (3) hematogenous infections of the gall-bladder and ducts; and (4) a spreading infection through the wall of the gall-bladder from an inflamed contiguous organ. Of these possibilities it is only the last two which take into serious consideration the actual infection of the wall of the gall-bladder, despite the fact that microscopic examinations of gall-bladders removed at operation, which we have studied, demonstrate that inflammatory changes are present not merely in the mucosa but also in the deeper layers, and that usually these changes are more pronounced in the deeper layers. The other two possibilities are concerned only with the entrance of organisms into the lumen of the gall-bladder, and they do not explain their entrance into its walls; they assume also that contact infection of the mucosa of the gall-bladder occurs easily and frequently. But there is no direct evidence to support the idea that cholecystitis occurs, except perhaps occasionally, merely as a result of the entrance of bacteria into the lumen of

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the gall-bladder; and there is much evidence, both clinical and experimental, that such infections are probably rare.

This evidence consists of a number of facts: (1) In experiments on ten dogs in which large amounts of pathogenic

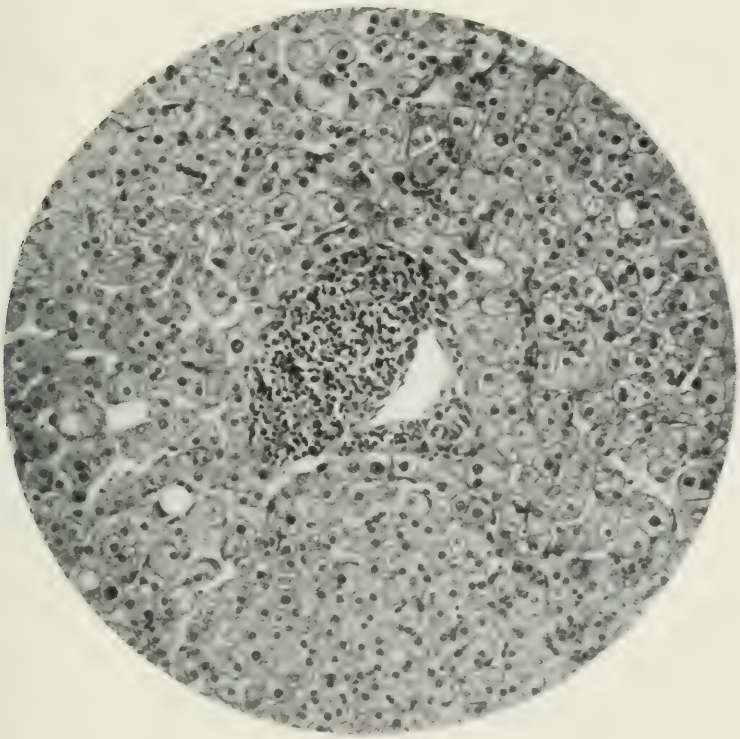

FIG. 1.—A common type of hepatitis occurring in association with a subacute cholecystitis. The periportal sheaths are densely infiltrated with leukocytes, chiefly of the polymorphonuclear variety. From a study of small pieces of liver removed at operation on cases of cholecystitis, either with or without stones, it has been noted that some degree of hepatitis is a constant accompaniment of cholecystitis.

colon bacilli were injected into the normal gall-bladder no cholecystitis was produced sufficient to be demonstrable by gross appearance or clinical signs, except in one case. In order to produce it with any degree of regularity it was necessary either

to obstruct the outflow from the gall-bladder by ligation of the cystic duct or injure the blood supply by ligation of the cystic artery. (2) Bacteria probably gain access to the gall-bladder by being carried down in the bile from the liver in most if not

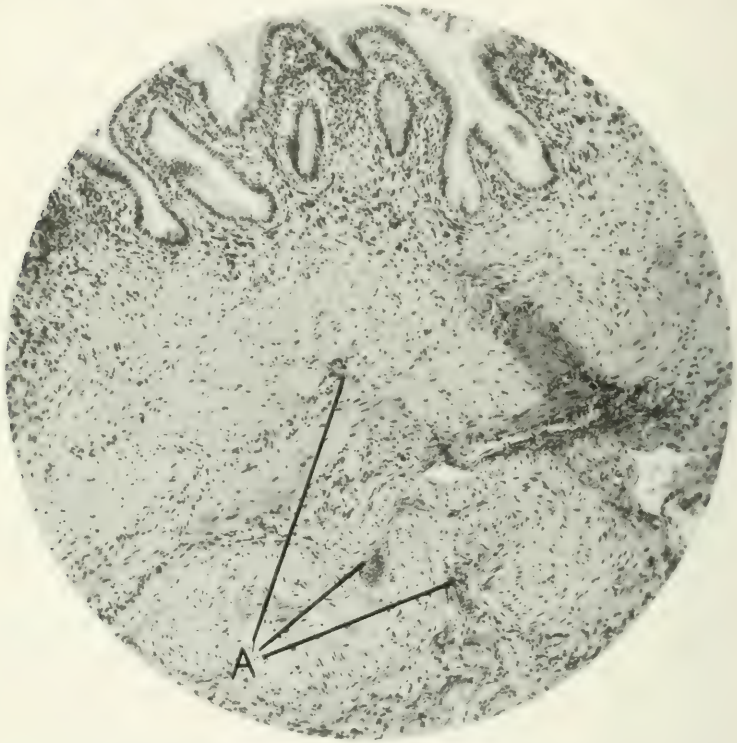

FIG. 2.—Gall-bladder removed at operation in case of cholecystitis without stones. Deep in the fibromuscular layers at a considerable distance from the mucosa are found collections of polymorphonuclear leukocytes, designated, for example, by lines marked *A*. These are usually much more conspicuous in the deeper layers than in the mucosa. Normally the mucosa contains large numbers of round cells (lymphocytes).

all cases of bacteriemia, yet cholecystitis is comparatively rare in such conditions. Rolleston[1] states that "in typhoid fever the bacilli are always present in the gall-bladder, but chole-

[1] Diseases of the Liver, Gall-bladder and Bile Ducts, p. 608.

cystitis is comparatively infrequent." (3) Theoretically the mere presence of bacteria within the lumen of the gall-bladder should be of no more significance in producing cholecystitis than the presence of bacteria in the urine should imply the presence of

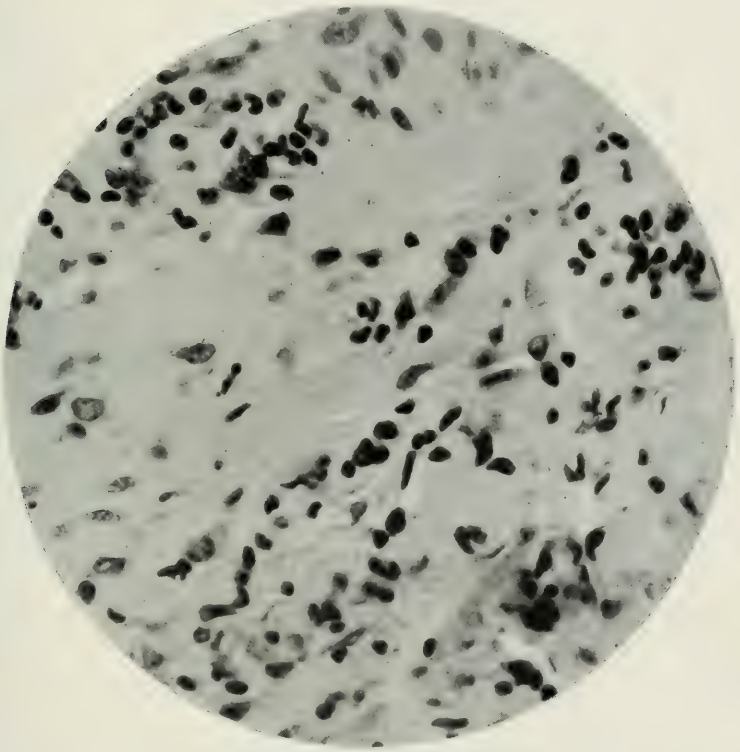

FIG. 3.—Higher magnification of the same gall-bladder as shown in Fig. 2. The leukocytic infiltration shown here is deep in the wall of the gall-bladder at a considerable distance from the mucosa.

an inflammation of the urinary bladder. It is well known that bacteria are frequently passed in the urine in large amounts without the coincidence of cystitis, especially in typhoid fever.

If the assumption is correct that the wall of the gall-bladder beneath the mucosa is involved in most cases of cholecystitis

then it would seem reasonable to discard the idea of the serious importance of contact infections of the mucosa by organisms within the bile, particularly in view of the evidence outlined above. How, then, may an infection originate in the wall? It

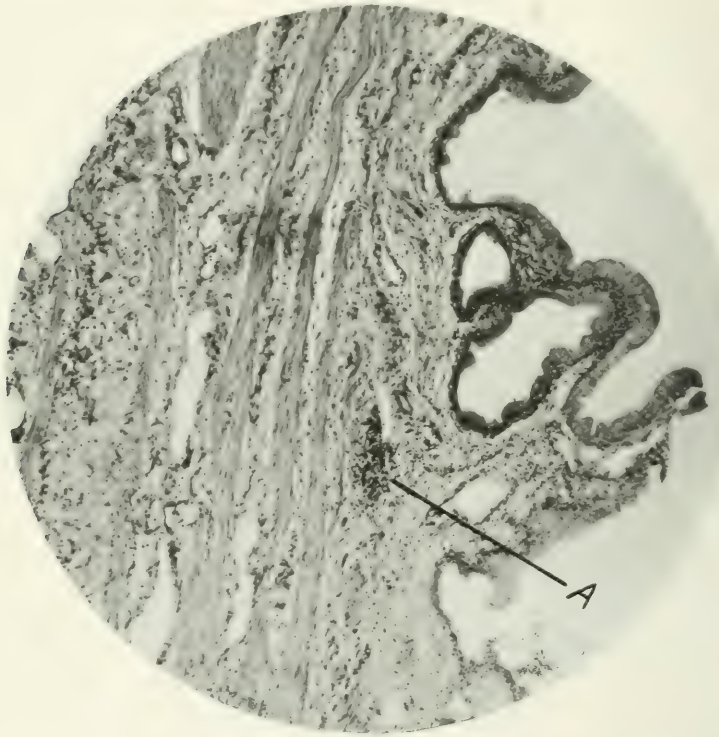

FIG. 4.—Another human gall-bladder with a rather typical collection of polymorphonuclear leukocytes at a distance from the mucosa. This is shown at the point marked *A*.

is obvious that in general only two routes are available for the transmission of organisms to the wall; one of these, the blood stream, has been particularly emphasized by Rosenow.[1] The

[1] The Etiology of Cholecystitis and Gall-stones and Their Production by the Intravenous Injection of Bacteria, Jour. Infect. Dis., 1916, xix, 527.

other is the lymph stream from the liver. In a previous article[1] we have called attention to the importance of this route not only in the pathogenesis of cholecystitis but also of its complications, such as pancreatitis, hepatitis and inflammation of the

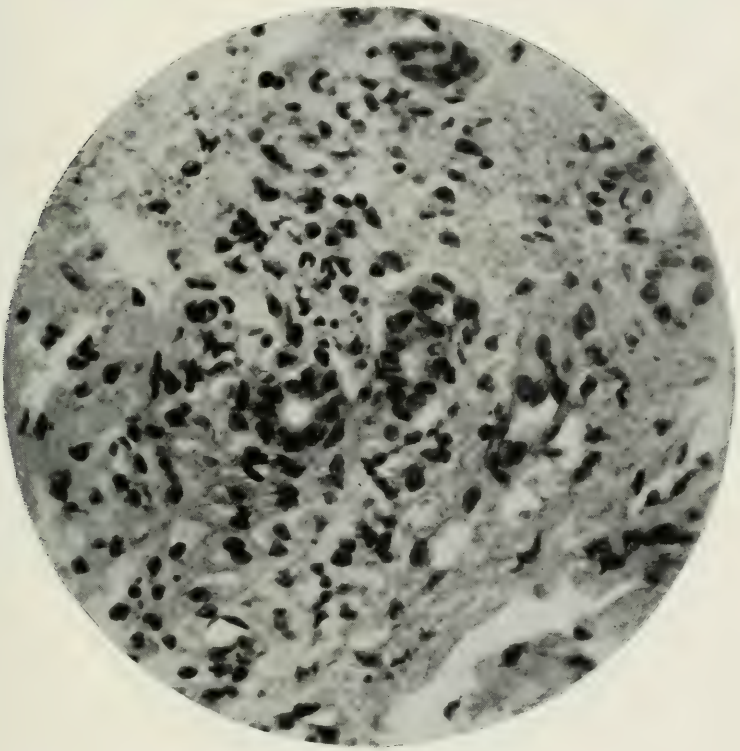

FIG. 5.—Experimental hepatitis in a dog produced by injection of the portal vein with streptococci. This section of the liver was made six days after the injection. Note the infiltration of polymorphonuclear leukocytes around the bile ducts.

common duct. Although the possibility of the lymphatic role in the pathogenesis of cholecystitis has been mentioned before,

[1] Peterman, M. G., Priest, W. S., Jr., and Graham, E. A.: The Association of Hepatitis with Experimental Cholecystitis and its Bearing on the Pathogenesis of Cholecystitis in the Human, Arch. Surg., 1921, ii, 92-115.

it is thought that the experiments carried out in this laboratory are the first which have brought direct evidence indicating that cholecystitis is not infrequently lymphogenous in origin.

The evidence, both experimental and clinical, seems to suggest strongly that perhaps in the majority of cases cholecystitis is

FIG. 6.—A gall-bladder from the same dog as shown in Fig. 5. The extensive leukocytic infiltration of the outer layers of the wall is well shown. The epithelium of the mucosa is intact and comparatively little changed.

produced secondarily to a hepatitis by lymphatic extension. This conclusion is based on a number of facts: (1) The almost constant occurrence of hepatitis with cholecystitis has been pointed out in a former article by one of us (Graham).[1] This

[1] Hepatitis: A Constant Accompaniment of Cholecystitis, Surg., Gynec. and Obst., 1918, xxvi, 521.

hepatitis is seen usually with the microscope to consist of an infiltration of the interlobular sheaths with leukocytes, chiefly of the polymorphonuclear variety, but in the more chronic cases there is observed a definite increase of connective tissue with a more or less extensive cirrhosis. (2) The well-recognized

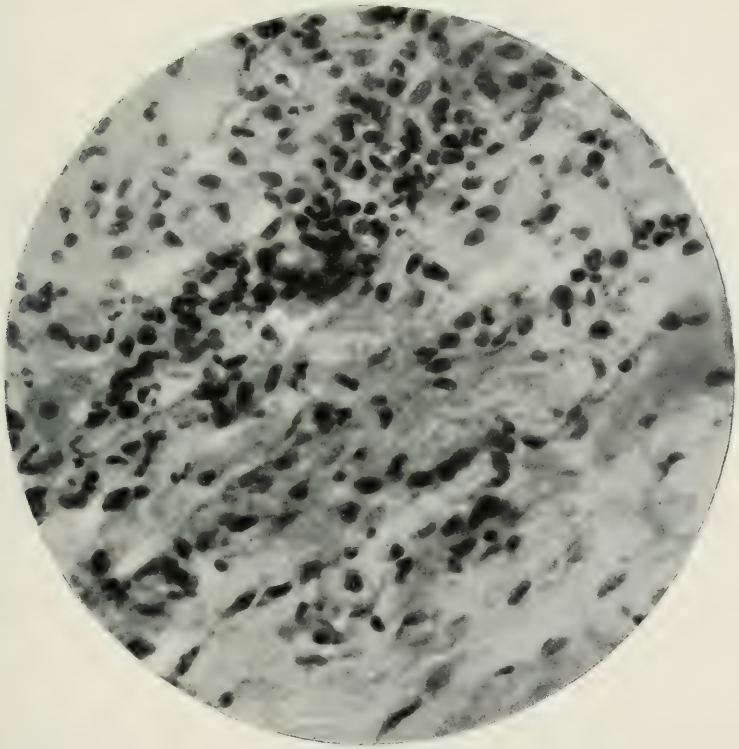

FIG. 7.—High-power photomicrograph of the wall of the gall-bladder shown in Fig. 6, taken at a distance from the mucosa, showing rather extensive infiltration with polymorphonuclear leukocytes.

association of lesions of the portal system (appendicitis, duodenal ulcer, etc.) with cholecystitis suggests that organisms are carried through the portal vein to the liver from these lesions and that a hepatitis occurs before the cholecystitis because there is no direct pathway between the appendix, for example, and

the gall-bladder. (3) By the direct injection of organisms (streptococci or staphylococci) into a radicle of the portal vein, as, for example, the appendix vein, it is possible to produce cholecystitis almost as a routine.[1] (4) There is no direct route

FIG. 8.—A gall-bladder from another experiment performed in the same manner as the one just preceding. This shows what seems to be a lymphatic vessel at the periphery of the gall-bladder containing polymorphonuclear leukocytes and presenting other evidence of acute inflammation.

by the blood stream by which organisms can be carried to the gall-bladder from the liver, since the cystic artery, which is the only source of arterial blood to the gall-bladder, is a branch of the hepatic. Organisms, therefore, to gain access to the gall-

[1] The details of these experiments will be reported later.

bladder from the liver *via* the blood stream would have to travel the circuit of the whole systemic circulation from the liver back through the celiac axis and the cystic artery. This would seem an unlikely occurrence in the majority of instances.

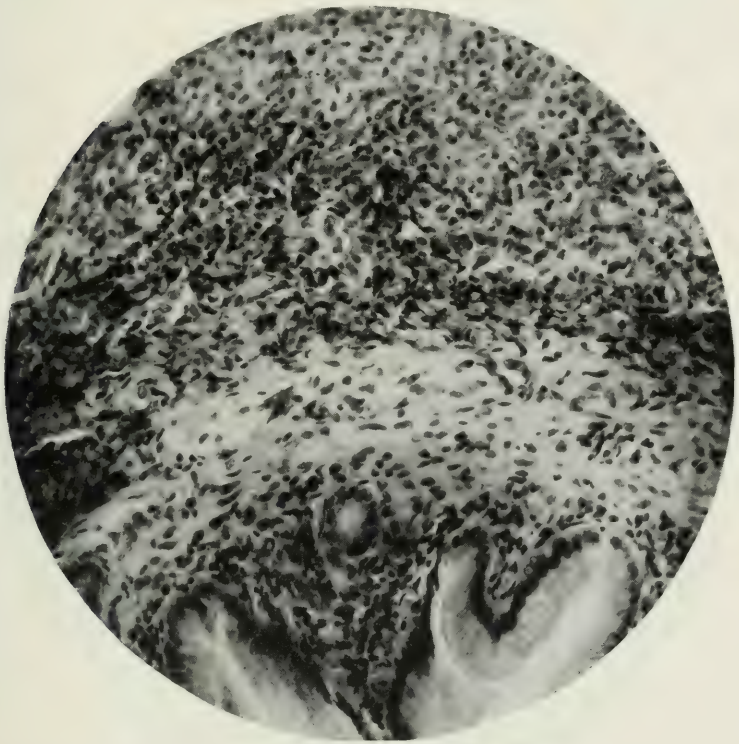

FIG. 9.—Extensive leukocytic infiltration of the wall of the common duct produced in a dog by injection of streptococci into the appendix vein. Hepatitis and cholecystitis were present also of the type already shown above.

The only possibility that remains, therefore, if we exclude contact infection of the mucosa by bacteria carried down in the bile, is the lymphatic route. The intimate lymphatic connection between the liver and the gall-bladder has been carefully worked out by Sudler,[1] and is shown in Fig. 12, which is taken from his

[1] The Architecture of the Gall-bladder, Bull. Johns Hopkins Hosp., 1901, xii, 126.

article. We have repeated some of Sudler's experiments and have confirmed his idea of the general arrangement and anastomosis of the hepatic and cholecystic lymphatics. The lymph vessels of the gall-bladder can be distended by injections of such a substance as Prussian blue into the portal vein. Conversely, also, injections made into the wall of the gall-bladder not only

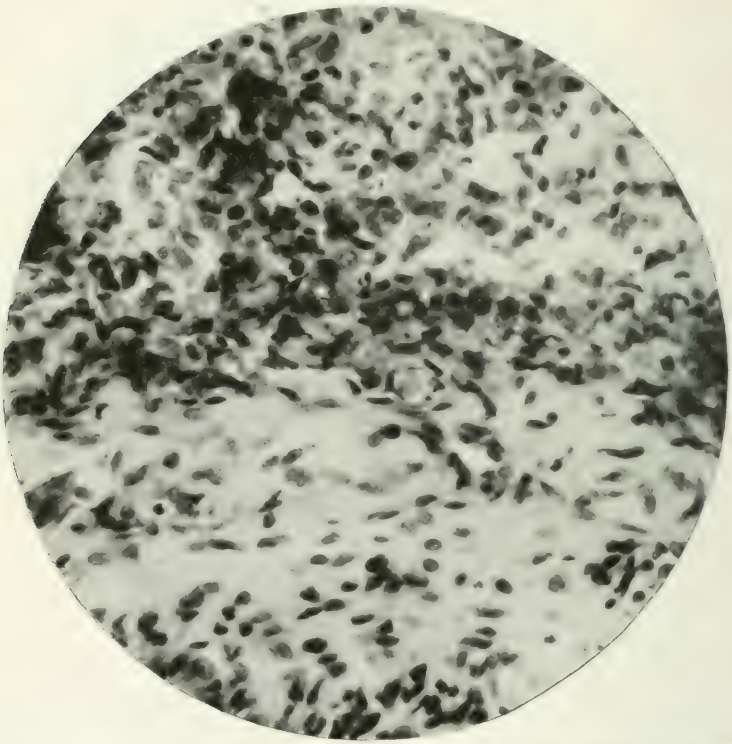

FIG. 10.—Higher power photograph of Fig. 9, to show the presence of both polymorphonuclear and mononuclear cells.

follow up the lymphatics into the liver in the interlobular sheaths but also may be seen to pass along the lymphatics of the common duct and into the pancreas.

It would seem, therefore, that infections of the biliary tract follow the same general rule as infections elsewhere in the body, in that they spread by way of the lymphatics. Given an infec-

tion of the portal system of sufficient intensity to produce a hepatitis, a cholecystitis might easily be expected to follow. Such a cholecystitis also, because of its lymphatic origin, would begin in the wall of the gall-bladder, and this hypothesis we believe agrees with the fact that microscopic examination of gall-bladders removed at operation shows that usually the

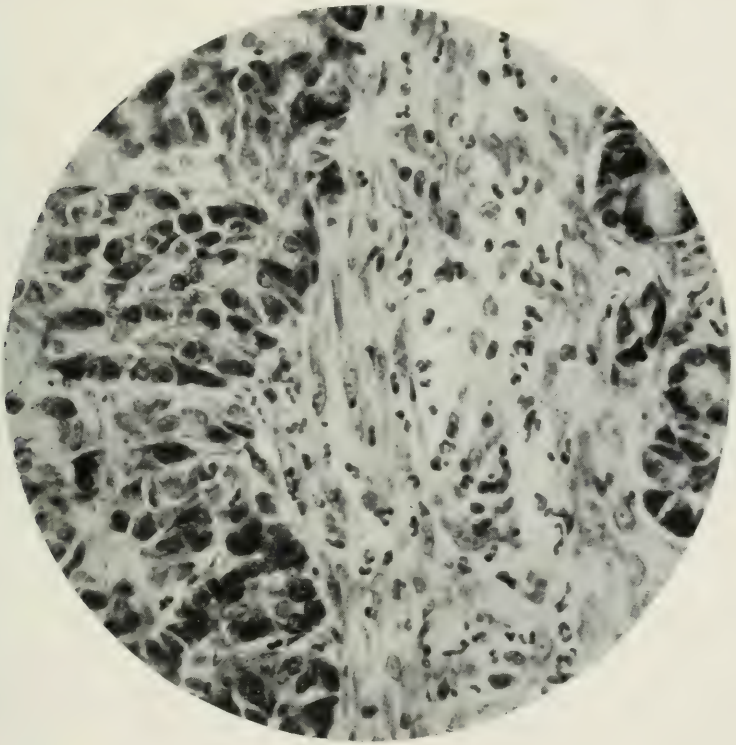

FIG. 11.—Interstitial pancreatitis in a dog six hours after an injection of a radicle of the portal vein with streptococci. Hepatitis, cholecystitis and choledochitis were also present.

inflammation is more pronounced in the wall than in the mucosa. We believe, furthermore, that too much attention is paid to the mucosa of the gall-bladder both by pathologists and surgeons and that therapeutic measures which are focussed on the mucosa alone, such as drainage or the stripping of the mucosa out of the

gall-bladder, are irrational and misdirected. The argument made here in favor of the lymphogenous origin of cholecystitis is not meant to imply that we think all cases have their origin in the lymphatic route. Doubtless some are hematogenous, some are certainly due to a spread of the infection from an adherent inflamed organ (as in duodenal ulcer occasionally), and

FIG. 12.—Anastomosis between the lymphatics of the gall-bladder and the liver (taken from Sudler). The extensive communication between the lymphatics of the liver and the gall-bladder is seen, particularly at the sides of the gall-bladder.

perhaps some, under exceptional circumstances, are even due to contact infection caused by organisms carried down in the bile. The possibility of the extension of the inflammation from the gall-bladder to the pancreas by way of the lymphatics has, of course, frequently been expressed. Maugeret[1] has written an excellent treatise on this subject.

[1] Cholecysto-Pancreatite, Thesis, Paris, G. Steinheil, 1908.

CHRONIC CHOLECYSTITIS WITHOUT STONES: DIAGNOSIS AND TREATMENT

By WILLY MEYER, M.D.
NEW YORK

Since patients with affections of the bile tract have become borderland cases, the operating surgeon has often been disappointed, when cutting down in the quiescent stage of the trouble upon the gall-bladder of a patient who had gone through a number of colicky attacks in the right side of the upper abdomen, to find a seemingly normal, soft, bluish and glistening organ, not harboring stones nor presenting any adhesions whatever.

No doubt many a surgeon, knowing and finding stomach and duodenum not to be the seat of the trouble, even today reluctantly removes the appendix in such a contingency and then, leaving the other organs intact, closes the abdomen; or, if the appendix had been removed at a previous operation, he considers the present interference a disappointing exploration and desists from further investigation.

But the patient does not get well.

Had, in a case like this, a more detailed examination been made, had *all* the diagnostic means which are at our disposal today been exhausted previous to the operative interference, the probabilities are that they would have given an indication for a procedure that would have been helpful to the patient.

I. Diagnosis.—On trying to analyze a patient's complaints of frequently recurring, or more permanent, pain in the upper abdomen, the physician whose advice is sought will

primarily think of disease of the stomach, duodenum, gall-bladder, or pancreas; secondarily also of the appendix and kidney.

A most carefully taken and minutely exhaustive history of the case and an equally thorough clinical examination will pave the way for a preliminary diagnosis. For an intelligent differentiation the laboratory then has to step in.

Determination of the quantitative and qualitative acidity of the gastric contents, string test with the use of the duodenal bucket, examination of the feces, Wassermann test and radiography with fluoroscopy will usually enable us to determine the presence or absence of gastric or duodenal affection.

Local tenderness and retention of part of the bismuth or barium meal at the end of twenty-four hours within the appendix, as shown by the *x*-rays, point to an affection of this organ. Its definite inclusion or exclusion in the diagnosis has gradually become of less importance in complicated troubles, since the majority of surgeons nowadays remove the organ in every case of abdominal incision, except the general condition of the patient forbids this addition, or adhesions bind the appendix down so firmly that it cannot be pulled into the wound. Even then, many of us, in order to take advantage of the anesthesia, quickly add a special incision in the right lower quadrant, and thus free the patient once for all from the possibility of appendicular disease.

The history, repeated urinary analysis, stereoscopic *x*-ray pictures, and, if indicated, cystoscopy with ureteral catheterization and pyelography will usually clear up the question as to the presence of surgical renal disease, particularly of a stone, in the greatest number of cases.

In the course of the last years the diagnosis of pancreatic affection has been greatly refined by the examination of the duodenal contents. If all three ferments—amylopsin, steapsin and trypsin—are found, and in sufficient quantities, serious pathologic changes can be excluded.

For the determination of the presence of gall-bladder disease

by means of laboratory findings, radiography, cholesterol test of the blood and examination of the duodenal contents with the help of the microscope as well as by chemical analysis and bacteriologic culturing are at our disposal today.

Radiography. At the last meeting of the American Gastro-enterological Association, at Atlantic City, May 3 and 4, 1920, A. W. George, of Boston, created quite a stir by maintaining that the healthy gall-bladder does not show on the plate under ordinary conditions; therefore, if the *x*-rays demonstrate a distinct outline of the gall-bladder, without showing the presence of gall-stones, such a gall-bladder is to be considered pathologic. He believes that this test may be relied upon in about 90 per cent of the cases.

This claim, if correct, would mean a valuable addition to our diagnostic capabilities as regards chronic cholecystitis without stones. In view of the importance of this matter, from a clinical point of view, I took occasion recently to obtain the opinion of five of the leading radiologists of New York City, as well as of two not residing in New York, and the present attitude of Dr. George himself. I requested their answers to the following questions:

1. Does the visualization of the gall-bladder on an *x*-ray plate mean a pathologic condition of the viscus?

2. In what percentage of cases do you think radiology able, at the present moment, to visualize stones in the bladder?

A reply was sent by everyone, for which I feel most grateful.

Looking the letters over with regard to question 1, it appears that of the eight specialists interrogated, three answered with a definite "yes"; three appear to lean toward answering the question in the affirmative; one replied "no"; one leans toward answering the question in the negative.

With reference to question 2, one considers it of relatively slight importance compared with question 1; he is inclined to "put the diagram of the adjacent hollow viscera first; a visibilized gall-bladder, particularly if altered in shape and density, second; and the direct detection of gall-stones, either positive or nega-

tive shadows, third. If all three of these are present, or even the first and second are present, he believes the *x*-ray diagnosis relatively certain."

One says: "The demonstration of gall-stones depends chiefly upon their amount of contained calcium. There are many ways of figuring percentages, but we are able to demonstrate only a small minority of gall-stones definitely and diagnostically." He adds as his belief "that in diagnosing disease either of the gall-bladder or appendix, the chief value of the *x*-rays is in excluding disease of the alimentary canal and genito-urinary tract."

One estimates the cases of gall-stones which are of sufficient density to produce characteristic shadows on the *x*-ray plate, under 25 per cent; 1, 32 per cent; 2, 50 to 60 per cent; 1, 85 to 90 per cent; one believes that visualization depends on the thickness of the patients, so much so that in those who are not extremely heavy practically all stones can be visualized; while he estimates a positive result in very fat patients equals 30 per cent.

Hope is expressed by one that pneumoperitoneum can be simplified, which would increase the positive percentage.

The conclusion we can draw from the foregoing is that, up to this moment, the assistance which the surgeons hope will be rendered by radiography in the fixation of the diagnosis of cholecystitis without stones, is in its evolution, promising however assurance of great improvement in the near future. And with regard to cholelithiasis it appears that at present radiography in the hands of experts may be relied upon as a means of helping to clear up the cases of recurring or permanent pain in the upper abdomen, as far as the gall-bladder carrying stones is concerned, likely in about 30 to 50 per cent of the cases.

I said advisedly "pain in the upper abdomen," and not pain in the right upper quadrant, because it is a fact that must be borne in mind that the subjective pain due to a diseased gall-bladder may be exceptionally confined to the left side of the upper abdomen, and may be noticed by the patient over the

left costal arch and the left side of the chest only, radiating from there to the left scapular region and shoulder, at no time involving the right side.

Since paying strict attention to this phenomenon for the last number of years, I have noted with increasing astonishment how frequently patients suffering from gall-stones complain of spontaneous pain in the region of the sigmoid, let us say in McBurney's point on the left, not seldom also tenderness at this point. This observation has been made particularly in emergency cases sent to the hospital with the tentative diagnosis of intestinal obstruction. It is only on closer examination that the surgeon will be able to make out that an inflammation of the gall-bladder is at the bottom of the trouble, that he has to deal with a reflex nerve phenomenon—paralytic ileus—and not an organic obstruction.

Cholesterol Test. Regarding the cholesterol test, I have made inquiries from a colleague who, for many years, has done original work along these lines. He writes that a blood cholesterol determination when properly interpreted "is of decided value in the diagnosis of cholelithiasis." He adds: "As with any test of this sort, it is paramount to realize its limitations."

Other authors think that the cholesterol test of the blood cannot be relied upon in the diagnosis of cholecystitis with stones, to say nothing of cholecystitis without stones.

In this uncertainty of laboratory assistance the analysis of the duodenal contents stepped in and has proven one of the greatest advances clinical medicine has made in the course of the last five or six years with reference to determining and segregating diseases of the stomach, duodenum, liver, gall-bladder and pancreas. Einhorn's duodenal tube has carried a bright light into the hitherto existing darkness as regards the more refined diagnosis of diseases of these organs, the same as cystoscopy and ureteral catheterization did literally some thirty years ago in connection with diseases of the urinary system. The aspiration, siphoning out and examination of the duodenal contents have pushed the recognition of the pathological condi-

tions of diseases in the upper abdomen as well as their treatment—a tremendous step forward. It is inspiring to note the increasing interest the subject awakens everywhere, particularly among the younger generation of medical men. There is hardly a man on the house-staff, I fancy, of any one of our larger hospitals, who has not become an expert in handling the duodenal tube and examining the collected fluid, who has not become an ardent disciple and co-worker in the evolution of this fascinating branch of medical and surgical diagnostics.

At the present moment physicians and surgeons are busy trying to determine the best and most useful and reliable method of collecting the duodenal contents.

When Einhorn and I commenced our combined work along these lines some five or six years ago, Einhorn had used his tube for diagnostic purposes for several years; in fact, he invented the method in 1909[1] for the purpose of diagnosing pancreatic disease. Later on he made use of it also in a therapeutic way, particularly for the treatment of gastric and duodenal ulcer. Since then its scope has been expanded steadily. I will but mention the healing of a duodenal fistula by means of a long duodenal (intestinal) tube (five cases on record so far); the use of the intestinal tube in diseases of the lower intestinal tract; duodenal flushing in the treatment of the so-called thrombo-angiitis obliterans and other cases of tobacco-smoke poisoning; duodenal feeding in operations on the mouth, pharynx, throat, esophagus, etc.

Regarding the collection of the duodenal contents, we consider it best to do it with the patient in the fasting condition. Einhorn's results of the examination of the contents thus obtained, covering a period of eleven years,[2] prove its advantages. With regard to the bacteriologic examination of the contents, Garbat has corroborated Einhorn's findings with regard to the best method of collection in every respect.[3] With the metal tip of the tube twenty-six to twenty-eight inches (about 66 to 71 cm.) from the incisor teeth, the duodenal contents are aspirated early on the morning following the introduction of the tube.

It is to be assumed that liver as well as gall-bladder—provided the cystic duct is not obstructed—are physiologically discharging some of their contents into the duodenum in spite of the fasting condition. If, exceptionally, no bile is found on aspiration, the injection of a few cubic centimeters of warm saline solution suffices to make it appear. The tip of the tube lies in waiting right in front of or somewhat below the papilla Vateri ready to receive the bile as it is physiologically discharged into the duodenum; suction from without then forces it into the barrel of the syringe. Green, yellow, yellow-green or black-green color and turbidity of the bile, the presence of pus corpuscles, particularly when in clumps, a small number of cholesterin crystals, and frequently numerous bacteria, point, we believe, to cholecystitis without stones; an increase in the quantity of these admixtures, particularly of the cholesterin crystals when they are very big in size and mixed with calcium or bilirubin crystals, to cholecystitis with stones.

If the radiograph and cholesterol test point in the same direction, such corroboration is, of course, gratifying.

Since the fall of 1919, particularly since the last meeting of the American Gastro-enterological Association at Atlantic City, May, 1920, the investigations of B. B. Vincent Lyon, of Philadelphia, have been brought more conspicuously before the medical profession. There is, I believe, hardly a center of medical investigation, hardly a hospital of standing in our states today, that is not actively and enthusiastically engaged in collecting and examining the differently colored bile as it is poured out of the common duct through Vater's papilla after the injection of 60 cc of a 25 per cent solution of sulphate of magnesium into the duodenum. Lyon was the first to suggest this procedure for the segregation of the bile from the different sources, on basis of Meltzer's theory that a concentrated solution of magnesium sulphate, when injected into the duodenum, would relax Oddi's sphincter and also lead to a contraction and emptying of the gall-bladder in consequence of the physiologic law of crossed innervation.

Lyon and his followers interpret the cycle of the changed color of the poured-out bile from dark yellow through black to light golden yellow, as representing the contents of the common duct, gall-bladder and hepatic duct—bile A, B and C. At the present moment it seems that this interpretation is not tenable. Einhorn's most interesting and convincing clinical and chemical investigations have shown that the dark-green, often black, bile (the so-called bile B), as caught by continued siphonage by the examining physician after the injection of the magnesium sulphate solution, *does not represent* the contents of the gall-bladder, but is discharged from the liver. It can also be produced by duodenal introduction of sulphate of sodium, peptone, glucose and some other substances. Moreover, the same phenomenon of the outflow of very dark bile after the injection of these different chemicals can also be observed in patients whose gall-bladder had been removed previously, thus distinctly showing that the dark bile does not emanate from the gall-bladder. It seems to be a secretion of the liver cells, pressed into increased activity by the absorption of the chemical substances from the duodenum by way of the venous system into the liver proper. Einhorn thinks that the dark color is probably due to the production of iron through the acute destruction of red blood corpuscles within the liver cells.[4]

A further clear proof for the contention that the so-called "B-bile" does not represent the contents of the gall-bladder is rendered by direct clinical observation. The bile poured out of the common duct after the 25 per cent magnesium sulphate instillation into the duodenum *always* shows the same dark brown-black color, whereas the gall-bladder content, if aspirated from the viscus during the operation, prior to cholecystectomy, is not in every instance dark-brown or black, but often green, green-yellow or greenish black, viz., of the same color as ascertained before operation, on collecting the duodenal contents with the patient in the fasting condition.

To repeat, the definite and, as it seems, correct conclusion arrived at by Einhorn is that the dark bile siphoned from the

duodenum with Lyon's test is not furnished by the gall-bladder alone, but also—if not principally—by the liver. Consequently the proper and advisable procedure for collecting the duodenal contents, in order to prove disease of the gall-bladder, is to do so with the patient in the fasting condition, and not after the liver has been artificially whipped into overwork.

As stated above, reliable data for the diagnosis of cholecystitis have been collected in this way, which many times were proven correct by subsequent operation.

II. Treatment. As in other diseases of the intra-abdominal organs of the borderline class the treatment may be either symptomatic or radical.

An individual of some means who suffers from chronic cholecystitis and is opposed to or for various reasons prevented from undergoing surgical interference, can visit Carlsbad, Vichy, French Lick or other spas of reputation, drink the waters there, and later at home continue the treatment, following a strict diet, often for lifetime, and taking care that he has regular bowel movements. Intermittent forced evacuation of the bile from the common duct by means of the instillation of the 25 per cent magnesium sulphate solution through the duodenal tube, and autovaccine treatment on basis of direct bacteriologic findings and cultures from the collected bile, may further contribute to the patient's well-being in the course of this conservative treatment. He may thus lead a fairly satisfactory existence and be able to hold in check the frequent, tormenting gastric hyperacidity. However, he is never sure of himself; at any moment, suddenly, unexpectedly and unprovokedly, a new attack may strike him. And the attacks due to cholecystitis without stones are often just as severe as those due to cholecystitis with stones, being likewise connected with fever, vomiting and general malaise. If such a patient gets tired at last and wants to be cured, or at least made as safe and comfortable as possible, the removal of the gall-bladder has to be advised.

A patient in moderate circumstances, afflicted with recurrent cholecystitis, should be differently advised and treated from the

start. Knowing that the absence of the gall-bladder is well borne by the human system, cholecystectomy is here clearly indicated. And the advice to have the gall-bladder removed may be given conscientiously and emphatically by the medical man, as it is an almost certain clinical fact that the chronic cholecystitis without stones very frequently is the precursor of the chronic cholecystitis with stones. Expressed in other words, a certain percentage of these patients will probably pass into the condition of cholelithiasis. In order to make them well and useful members of family and community, from an economic point of view, cholecystectomy should be advised.

With the examination of the duodenal contents added to the series of required laboratory examinations in every chronic case of abdominal pain, above as well as below the umbilicus, the additional presence of a cholecystitis will often be detected, where appendicitis alone would be diagnosed by those who do not practice the method.

Having recognized a chronic, recurrent gall-bladder inflammation to be present besides a chronic appendicitis, and, perhaps, the principal cause of the symptoms complained of, we do not proceed by the unsatisfactory method, still practised by many, of cutting down upon the appendix and then palpating the gall-bladder to find out whether or not it contains stones, and if so, remove it only then. With the refined diagnosis before us, it will become our duty to cut down upon the gall-bladder first, and then pull up into the wound the cecum for the removal of the appendix. In this way we shall be enabled to inspect and palpate incidentally any other organ of the upper as well as lower abdominal cavity. With such preoperative, thorough examination and corresponding operation a much greater percentage of patients with chronic, recurring abdominal disease will get well with the help of *one* operation, instead of two or more.

Personal work done in this direction and on this basis, in conjunction with Dr. Einhorn, for the last five or six years, justifies the following:

III. Summary.[5] 1. Aspiration of bile from the living gall-bladder during operation having shown the same stigmata as that collected from the duodenum in the fasting condition, before operation, definite proof has been rendered that the examination of the duodenal contents obtained by the duodenal tube, with the patient in the fasting condition, deserves confidence and is to be considered a reliable procedure.

It is no hardship for anyone to swallow the tube; with some little persuasion the most sensitive patients will permit even a repetition of the procedure should this become necessary.

2. A glistening, bluish, soft and non-adherent gall-bladder found during operation may be diseased and harbor pathologic organisms within its walls.[6] These organisms leave their place of habitation at times and enter the gall-bladder contents, infecting the bile stored within, although this bile has left the liver and reached the gall-bladder in a sterile condition. Recurrent attacks of inflammation, varying in severity according to the degree of virulence of the bacteria, will be the immediate consequence.

3. The bile within the gall-bladder, though discolored and turbid, is often found sterile. This is explained by the bactericidal action of the bile as such, and the but occasional presence of active bacteria within the bile. Aspiration of a certain quantity of bile at a given moment during the operation may just strike a sterile fluid, which, at other times, may contain bacteria.[7]

4. In view of the fact that bacteria are frequently found in the center of gall-stones, we can understand how it happens that cholecystitis without stones is the precursor of cholecystitis with stones.

Cholecystectomy in cases of cholecystitis without stones, therefore, clearly represents a prophylactic operation in many instances, and is of particular benefit to those who must get well in order to be able to earn their living.

5. It has been shown that the black fluid furnished by the Meltzer-Lyon test, the so-called B-bile, as collected from the

duodenum by siphonage through the duodenal tube, after the instillation of a 25 per cent solution of magnesium sulphate, is mostly a liver product, representing the immediate effect of absorption of the injected substance upon the liver cells. The bile contained in the gall-bladder seems to play no important diagnostic rôle in the cycle of secretions, though frequently some of it will be admixed to the so-called B-bile.

Repeatedly direct visual inspection of the gall-bladder during operation, with the tube in place and the instillation of a 25 per cent magnesium sulphate solution into the duodenum, has failed to show the slightest physical contraction of the viscus, the latter having been under closest observation of the author and his associates for more than twenty minutes.[9]

6. The Meltzer-Lyon test has therefore little, if any, value in diagnostic respects, but it promises to be of great importance in the *treatment* of diseases of the biliary system.

7. Chronic cholecystitis without stones is a much more frequent disease than has been heretofore believed. Clinically, it seems to take the same place in the upper abdomen as chronic appendicitis does in the lower.

Chronic appendicitis, on account of its treacherousness and eventual danger to life, when suddenly flaring up, is generally conceded to be a surgical disease requiring operation. It is no longer a disease of the borderland.

Chronic cholecystitis, as mentioned above, still belongs to the borderland. Though not as treacherous and deadly in its sudden attacks as the inflammation of the appendix may be, it nevertheless often undermines the patient's health and should, therefore, as a rule, require the removal of the gall-bladder.

If only drained temporarily, such a gall-bladder may become the seat of a recurrent inflammation at any time and without any known provocation; it may also harbor stones at a later date. A patient thus afflicted who will not or cannot submit to cholecystectomy, may then pass into a state of chronic invalidism. His gall-bladder represents the same type of infected focus from which absorption takes place intermittently—auto-

intoxication—as the tonsils, suppurating teeth and intestinal stasis do.

Cholecystectomy represents the radical cure, and should be advised after the laboratory analysis, outlined before, has established the refined diagnosis. It appears that cholecystectomy is as well borne by the human system as appendectomy. The mortality of the operation, done in the interval, is almost as negligible as that of appendectomy in the interval stage. Here, too, we may expect that 100 per cent of the patients recover; some extraordinary and unforeseen something, beyond the surgeon's control, would have to happen if the patient did not recover.

9. Regarding technic, the Perthes incision is favored by the author in the strong male patient in order to restore the abdominal wall to absolute perfection.

He also believes it to be to the best interest of the patient, in the course of cholecystectomy, to advance from the gall-bladder fundus toward the common duct. This will enable the surgeon to meet possible anatomical variations as regards the bloodvessel and cystic duct arrangement (Eisendrath), and will further enable him always to place the ligature of the cystic duct close to the common, thus preventing the later formation of a miniature gall-bladder, as some authors claim to have observed after extirpation.

Temporary drainage of the abdominal cavity after cholecystectomy is considered an absolute necessity. Air-tight suture of the peritoneal sac means taking chances with the patient's life, as the ligature of the cystic duct may give way for unknown reasons a few days after operation, and the leakage of infected bile then cause peritonitis and death.

10. The principal technical advantage accruing from the preliminary more refined diagnosis of diseases of the bile tract, in the presence of gall-bladder disease *combined* with appendicular disease, is the indication derived from it with reference to the place of the incision. Knowing *before* operation that the bile is pathologic, we will cut down on the gall-bladder and pull up

the cecum with the appendix, instead of cutting down on the appendix and then palpating the gall-bladder region, as done heretofore. In the latter instance the surgeon can but determine the presence or absence of calculi in the gall-bladder; he cannot feel pericystic adhesions, omental attachments, etc.

11. Inspection and palpation of the gall-bladder during operation must cease to render the only indication for cholecystectomy. The *quality* of the bile, determined systematically, with the help of the duodenal tube *previous* to operation, and, if desired, the condition of the bile aspirated from the gall-bladder *during* the operation, must guide the surgeon in his decision of where to incise and what to excise.

Many important questions, naturally, still remain unsolved and require further study, for instance:

Is the color of the normal bile always golden yellow; in other words, does green or brown bile in a gall-bladder, or a mixed color, always indicate pathology?

May the wall of a gall-bladder contain microörganisms and the viscus still be considered non-pathologic? In other words, does the presence of bacteria in the wall of a macroscopically seemingly healthy gall-bladder *always* represent the first stage of the cycle of the pathologic events up to the eventual formation of gall-stones?

Do the bacteria reach the viscus from the duodenum, or by way of the arterial blood stream, or the lymph current?

IV. Final Results. A final follow-up of results obtained so far in our series of cases of cholecystectomy done for chronic cholecystitis without stones was started, but it has been impossible to complete it and, therefore, cannot be embodied in this paper.

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6. Brown, R. O. (Rochester, Minn.): A Study of the Etiology of Cholecystitis and its Production by the Injection of Streptococci, Arch. Int. Med., February 15, 1919, No. 2, xxiii, 185. See also Einhorn, Max, and Meyer, Willy: New York Med. Record, loc. cit.

7. In order to obtain positive results of examination as often as possible, it is necessary to spread the bile on a culture medium immediately after aspiration from the bladder, or after incision of the gall-bladder in its aseptic condition directly after extirpation. It is further advisable to transversely divide the extirpated bladder which did not contain stones and place half of it into a 5 per cent formalin solution immediately after extirpation for pathologic examination, and have the other half used without delay for tissue culture growth at the laboratory.

8. Aschoff, L., and Bacmeister, A.: Die Cholelithiasis, Jena, G. Fischer, 1909.

9. Of course this kind of observation is not convincing, because physiologic conditions are changed. A gall-bladder may act differently with the abdomen closed.

DISCUSSION ON THE PAPERS OF DRS. ARCHIBALD, SISTRUNK, OCHSNER, DEAVER, GRAHAM AND MEYER.

Dr. William J. Mayo, Rochester, Minn.:

I have been interested in the papers dealing with infections of the gall-bladder and pancreas. One point might be brought out in connection with Dr. Graham's excellent paper, and that is the part the lymphatics play on the course of infection in the liver, gall-bladder and pancreas. The portal pressure is 30 and the general circulation in the hepatic artery is 130; the pressure of the blood from the hepatic artery on the portal circulation is the main factor in the circulation in the liver. The hepatic blood-pressure in connection with cirrhosis of the liver is important. If the hepatic artery is tied carnivorous animals usually have no serious trouble, but herbivorous animals usually die.

I agree with Dr. Meyer's views on cholecystectomy. Dr. Lilienthal was one of the earliest to call attention to the value of cholecystectomy as compared with cholecystostomy. In regard to prolonged drainage of the gall-bladder, if a gall-bladder is drained, say from seven to ten days, until clear normal bile is secured, provided there is no obstruction to flow into the duodenum, the tube can be removed and the patient will recover at once. If the drains are left in longer the bile begins to turn green, the patient becomes depressed and several weeks may elapse before the bile again becomes sufficiently normal to permit withdrawal of the drains.

Considering the question of the pancreas from an operative standpoint, on many occasions in resections of the stomach for carcinoma I have removed considerable superficial portions of the pancreas at points of attachment without after-disturbance. I have removed tumors from the body of the pancreas and sutured the ends together successfully. The pancreas withstands operation well.

Dr. John H. Gibbon, Philadelphia:

I was very much interested in the paper of Dr. Sistrunk on pancreatic stones and the difficulties in determining the presence of these stones. It seems to me that if there is a pathologic condition in the abdomen which is found more frequently at autopsy than at operation in these days of the "open door," it is a sad commentary on our diagnostic ability. I cannot see how we are to get along much better than we do now in the diagnosis of pancreatic stones, but I should like to ask one question, and that is whether in these cases that he has

referred to the *x*-rays have shown these stones. There is no doubt about it that the *x*-rays are showing up more and more gall-stones, and I am anxious to know whether pancreatic stones are shown.

One other point in regard to these cases that were reported that I think is worthy of our attention, and that was the suggestion in the paper that these stones that are in the lower part of the duct be removed through the duodenum. I do not think that this method is used universally. Years ago it was very prevalent in France, and I believe that the transduodenal route is the route by which to remove stones from the lower portion of the common duct and the ampulla. I have done this several times and have been astounded at the ease with which it is done as compared with the removal of stones through an incision in the common duct. Moreover, I believe the removal in this way will reduce the danger of forcing infected material into the pancreatic duct.

There is one other point in regard to Dr. Sistrunk's paper, and that is the quotation from Osler in regard to the occurrence of cholecystitis during typhoid fever. I do not think that the incident of cholecystitis during typhoid has any relation at all to the infection of the gall-bladder and the subsequent development of stones, because we all know that typhoid infection is a very insidious one and there may be no evidence of cholecystitis during the course of typhoid fever; but I have been surprised to find how many patients operated upon for gall-stones have had typhoid fever.

I recall one patient of mine, on whom I operated, who had typhoid fever thirty-one years ago and typhoid bacilli were found in the stones. I have another case in which the typhoid bacillus was found eighteen years after, and a third case which was most interesting: A woman who said that she had never had typhoid; we took stones out of her gall-bladder, and a few days after the operation she had a perfectly typical typhoid fever with a positive Widal reaction.

I agree with Dr. Deaver about the gastrologists, but I think that Lyon's work is of distinct practical value to the surgeon. I believe that Lyon is capable of showing us the bile from the liver and from the gall-bladder, and just as a positive *x*-ray plate showing stones is of great diagnostic value and a negative film is of no value, so I believe that if these gastrologists can show us by stimulating the gall-bladder that the bile does contain organisms, then I think it is of great value. When it comes to treatment by this method I think it is overdone, and I agree with Dr. Deaver absolutely. From the diagnostic point of view I think these men can help us a great deal, and we should pay attention to their work.

Dr. Leonard Freeman, Denver, Col.:

It occurs to me in this discussion that it should be emphasized that there is such a thing as marked gall-bladder trouble without infection; in other words, what is know as "stasis gall-bladder." Attention was called to this in 1909 by Aschoff and more recently in an article by Schmieden. These stasis gall-bladders are very definite things, and it so happens that in my limited experience I have had occasion to observe a number of them. The stasis is produced in several different ways: For instance, it may be caused by an anomalous Heister valve in the cystic duct or by the cystic duct being attached very high at the side of the gall-bladder. The result is that when the gall-bladder becomes overdistended with bile its proximal end presses against the cystic duct and shuts it off. There may also be other anatomic reasons. I think everyone in operating upon gall-bladders has found that if he tries to press out the bile suddenly it will not go out. If he presses on the gall-bladder slowly then the bile goes out of the cystic duct without trouble. In other words, overdistention and spasm may cause temporary stasis and colic.

In this condition Aschoff insists that gall-stones are formed without infection and that they represent 27 per cent of all gall-stones. These stones differ from ordinary gall-stones in that they are formed by crystalization from a center while the ordinary gall-stones are formed in concentric layers. Sometimes a combination exists.

In those gall-bladders affected with stasis the symptoms may be extremely pronounced. I venture to say that it sometimes may be impossible to tell the symptoms of a stasis gall-bladder from the symptoms of an infected gall-bladder or a gall-bladder with stones. There may be no change in appearance, so there is great danger of overlooking it, and yet the symptoms may be pronounced. In several cases that I have operated upon the colic was extreme, and yet after the operation all of the symptoms disappeared. It would seem that we should pay more attention to this preinflammatory stage and recognize that it is more frequent than we have supposed.

Dr. Miles F. Porter, Fort Wayne, Ind.:

In reference to the diagnostic value of bacteria in the bile in cholecystitis, we compared this to the Arab who had a stone from Mt. Ararat, and offered this as proof that the Bible story of the flood was correct.

I know of operations that have been based upon these findings wherein the subsequent examination of the gall-bladder revealed no pathologic condition whatever.

There is another point that suggests itself, and that is the title of

Dr. Archibald's paper, which suggests that a large number of cases of pancreatitis are cases that originate from an infection primarily in the liver. If we take this into consideration plus the much better results as evidenced by Dr. Deaver from prolonged drainage I think we can arrive at an explanation for the difference between those who would remove all gall-bladders and those who only remove some. A careful study of my own work shows results less unfavorable to cholecystostomy than does the work of some others. There is only one explanation that I can give, and that is the drainage is prolonged, as suggested by Dr. Deaver. I believe that we will by and by arrive at more definite data upon which to base our conclusions as to the best method of treating these cases, whether by cholecystectomy, drainage of the common duct or drainage through the gall-bladder. I would suggest again that there is room for making a serious mistake by attaching too much attention to bacterially infected bile. It does not necessarily mean a cholecystitis.

Dr. Stanley Stillman, San Francisco, Calif.:

In discussing Dr. Deaver's paper, I may say that in my younger days I had two cases that taught me a lesson. Both cases had a palpable tumor and were jaundiced. Both had stones in the gall-bladder and the common duct. In both the head of the pancreas was very large, hard and nodular. In both the gall-bladder and duct were drained, as was the custom at that time, but an unfavorable prognosis was given, as I was sure they both had cancer of the head of the pancreas, never at that time having heard of chronic pancreatitis. However, the patients kept on living, year after year, and may be living yet, much to my surprise and subsequent enlightenment, and are good examples of chronic pancreatitis that recovered completely after gall-bladder drainage, as Dr. Deaver has indicated.

In reference to Dr. Graham's very satisfactory paper I should like to mention a case that I think is quite confirmatory of his views from a clinical standpoint.

The patient was a woman, the wife of a physician, who had had her gall-bladder drained for symptoms that were characteristically those of a cholecystitis. The drainage was followed by relief of her symptoms for a period of several months, and then they all returned as bad as ever. A second time the gall-bladder was drained, and again she had temporary relief, to be followed again in a few months by a return of the original symptoms.

She then came to San Francisco and her husband insisted that the gall-bladder should be removed, and asked me to do it.

On opening through the old cicatrices we were surprised to find no adhesions, and the cicatrices in the fundus of the gall-bladder were hardly perceptible. The gall-bladder was apparently normal, perhaps a little thickened at its base. I was loth to remove it, but the doctor insisted, having been present at both the preceding operations. The removal was accomplished without difficulty, and I thought of closing without drainage, but finally inserted a tube.

The operation was immediately followed by peritonitis and by infection of the wound, and later by obstruction of the bowel, necessitating a right-sided colostomy. The patient entirely recovered and has been perfectly well for the past five years or more, and at no time has she had any suggestion of her original symptoms.

There was no escape of bile at the operation, and the bile contained no streptococci; but staphylococci were found in the wall of the gall-bladder at its base and the abdominal infection was a streptococcus infection. I am very sure that the general infection came from the stump of the cystic duct, which was not cauterized, and that the cholecystitis was confined to the walls of the gall-bladder and duct, and I think the indication is very strong that the infection of the walls occurred through the lymphatic system, as suggested by Dr. Graham.

Dr. D. F. Jones, Boston, Mass.:

After what has been written about cholecystitis without stones, up to the present time, it would seem to be necessary to remove the gall-bladder when we open the abdomen, for the gall-bladder does not seem to be subject to the same laws as other organs, and inflammation which causes symptoms sufficient to demand an operation does not cause any changes that can easily be made out clinically.

We may have a valve action at the entrance of the cystic duct which causes stasis and symptoms for which it is necessary to operate, yet these conditions have no effect upon the gall-bladder itself. A valve action of the ureter at its entrance into the pelvis or a narrowing of the ureter sufficient to cause symptoms will cause dilatation of the pelvis which can easily be made out clinically.

An inflammation of the pelvis of the kidney will in a very short time cause sufficient thickening to be easily made out clinically, yet a cholecystitis causing symptoms which demand operation cannot in many cases be detected except under the miscroscope.

Dr. Alexis V. Moschcowitz, New York:

As a general rule it may be stated that it is safest to agree with Dr. Deaver. At the same time I should like to place on record a case that I treated, to all intents and purposes, according to the advice just

given by Dr. Deaver. The patient was one whom I operated on for a subacute pancreatitis. Having in mind the work of Dr. Deaver I drained the gall-bladder for several weeks, and while the gall-bladder was still draining the patient suddenly developed a hyperacute pancreatitis, to which she succumbed.

Autopsy revealed an enormous hemorrhage into the pancreas and retroperitoneal tissue.

Dr. Arthur Dean Bevan, Chicago.

I have been very much instructed by the symposium on the pancreas this morning. I think the work of Dr. Graham is very illuminating, and we should look forward in the future to more definite information in regard to infections of the liver and biliary tract than we have at present, and along the lines that Dr. Graham has brought out.

I want to say a word in regard to Dr. Deaver's paper: I have been very much impressed with the value of what he calls external drainage by cholecystostomy as compared with what might be called internal drainage as represented by cholecystenterostomy. I think there is little doubt, certainly in my own mind, that the external drainage is much the safer procedure. One of my assistants recently has been doing some work on dogs, and his work shows how constantly cholecystenterostomy produces infection of the liver. Of course, this is not nearly as liable to occur if the drainage is external.

I agree with Dr. Deaver, too, when there is a definite indication for drainage in a chronic interstitial pancreatitis provision should be made for drainage over a considerable period. Where the common duct is used a practical plan is the use of the T-tube, which can be maintained for a long time. I have employed it in a number of cases for more than a year. There is, however, one singular thing about some cases of chronic interstitial pancreatitis, and that is that a number of them get well without any drainage at all.

My experience has been gained in a series of cases in which a definite diagnosis from the findings at the exploratory operation was carcinoma of the pancreas. In such cases I have closed the incision completely, informing the friends and family that we were sorry that the patient had carcinoma of the pancreas, and would come to a fatal termination within a short time. In three cases in one year, instead of a fatal termination, they went on to a permanent recovery. Undoubtedly they must have been cases of interstitial pancreatitis.

Dr. Meyer has brought up one of the most important subjects, chronic cholecystitis. I think this is one that should be handled with good judgment. I have had many such cases in which I could find no clinical reason for doing an operation. We have no more reason for

operating on these cases because of the mere evidence of the presence of bacteria in the bile obtained from the duodenum than we have for operating on a man's tongue because he has hemolytic streptococcus on it. Just at present we hear of a great many medical men who are operating on all tonsils because they find the hemolytic streptococcus on them, but they have not got so far as to amputate the tongue for the same reason. I do feel that this is a very dangerous topic, this matter of chronic cholecystitis in what appears to be a normal gall-bladder. My own conception is that in the vast majority of these cases, where after a careful examination with your colleague in internal medicine you have arrived at a diagnosis of probable gall-bladder infection and at exploration you find a normal gall-bladder, you can be certain the chances are ten to one that it is normal and does not require removal. I think it is a very serious mistake to remove the gall-bladder in such conditions. Many of these cases are simply mistakes in diagnosis—the symptoms are due to neurosis or a lesion of some other organ and the patient is not benefited by the removal of the gall-bladder.

Dr. M. L. Harris, Chicago, Ill.:

We have all met with cases presenting clinical evidence of diseases in the upper right quadrant of the abdomen which we have diagnosed as trouble with the gall-bladder or gall-tract, in which on operation we have failed to find as much pathology in the gall-bladder as we had expected.

I want to call attention to three conditions which may help explain some of the cases belonging to the group to which Dr. Meyer has called attention. (Blackboard demonstration.)

The first condition is one in which there is a movable kidney which moves about a transverse axis. The kidney moves in a forward direction, and when the patient is put in the proper position the kidney will lie against the anterior abdominal wall.

The second condition is where we have a movable kidney in an up-and-down direction with a persistence of the hepatorenal fold, a distinct peritoneal fold passing from the kidney to the gall-bladder, with a movable gall-bladder.

The third condition is where we have a movable liver in a thin woman. The liver slips forward and downward and kinks the cystic duct.

These conditions all bring up distinct symptoms in the upper right quadrant.

Dr. Joseph A. Blake, New York:

I have been very much interested in the apparently contradictory statements in regard to infection of the gall-bladder. It seems to me

that we are paying too much attention to obstruction of the cystic duct. We have had a great deal of evidence brought forward that the infection of this organ may be primarily interstitial and not arising from obstruction of the duct or from contamination of its contents. We know very well that an organ that is inflamed does not contract properly, and why may it not be that in interstitial inflammation of this organ the ability of the organ to contract and empty itself is interfered with. This would lead to stasis, and we know where there is stasis that the contents may become contaminated and infected.

In regard to drainage, Dr. Mayo speaks of what has been a hobby of mine for years. I should not think of keeping a drain in a gall-bladder or in the common duct when I have evidence that the bile could pass through into the duodenum through its natural channel. We have a great deal of evidence to prove that the introduction of drains and continuing drainage not only provokes but continues infection. Perhaps no surer proof of this can be found than in infection of the joints. We know very well that in infected knee-joints if one attempts to cure them by drains passing into the synovia, and particularly if through the joints, the joints will be lost.

Dr. Howard Lilienthal, New York:

The statement of Dr. Jones that continuous internal obstruction will cause distention of the gall-bladder cannot be confirmed by me. If the obstruction is inside the gall-bladder, and is due to stones, it will cause contraction of the gall-bladder and thickening of its walls. We all know of the enormously distended gall-bladders in carcinoma of the head of the pancreas. The presence of the diverticulum, which I think is known as Hartmann's pouch, is an added reservoir for bad drainage and infection. A gall-bladder with this deformity should be removed.

A gall-bladder cannot always be properly explored without mobilizing it.

I have been fortunate enough in relieving patients of severe symptoms of gall-bladder disease in which no stones were present but in which I was able to demonstrate a stricture of the cystic duct after the viscus had been removed.

One word about the Lyon test: At Mt Sinai Hospital, New York, Dr. Crohn has made numerous experiments in my service. The findings were seldom satisfactory and the test has been given up. I do not think this is quite fair. Hartmann's pouch and the neck of the gall-bladder are most likely to contain purulent bacteria in large numbers. The single test by the Lyon method is not sufficient, and

it should be made at least twice at an interval of one or more days. They usually make this test soon after the patients enter the hospital It should be repeated just before operation.

Dr. Emmet Rixford, San Francisco, Calif.:

It takes a good deal of courage to say a word on the other side, but my only excuse is that I am in trouble about prolonged drainage of bile. Dr. Deaver tells us that prolonged drainage is the thing. I have nothing better to offer than drainage, and that is why I am in trouble. I have had a number of cases in which prolonged drainage has been made, and those cases, several of them, have done badly, have become weak, and have persistently vomited and become more and more emaciated. In one man whose gall-bladder was removed for stone, drainage persisted for weeks after the tube was removed. The man went down and down until he was all but comatose. In desperation I opened the abdomen and sewed up the duct. The patient ceased vomiting from that moment; the next day he was definitely on the mend, he took more and more food and had not a setback thereafter. He is perfectly well today, two years after the operation.

In another case I had almost the identical thing occur when I sewed up the duct. The patient stopped vomiting, began to take food and did wonderfully well for four days, and then the chromicized catgut with which I had sewed the duct was absorbed. Perhaps I should have used silk, but I was afraid to leave an unabsorbable suture in the wall of the duct for fear it would become the nucleus of more gall-stones. In the second operation I found an entirely new set of gall-stones in the duct anyway, but they were so soft that they could scarcely be called stones; a touch of the finger would crush them. I removed all of these as far as I knew and went so far as to introduce a catheter into the hepatic duct and washed out another stone from up in the hepatic duct, but all to no avail. Drainage continued and the patient succumbed. Both of the patients that I have described lost in addition to their bile their pancreatic fluids, a fact which may be of considerable importance in the premises.

Dr. J. J. Buchanan, Pittsburgh, Pa.:

Those of us who have spent the large part of our surgical lives in draining gall-bladders ask ourselves the question whether bile is necessary for the human being. We see patients by the hundreds draining bile for weeks, and they do well; they do not seem to mind it. We catch it in our rubber bags and the patients get along well and the opening closes. Then we get a patient occasionally with stone in the common duct. We drain this common duct and the patient does well

up to a certain time, as has been pointed out, and then the patient, as described by Dr. Rixford, vomits a little, loses his appetite, becomes weak and commences to shrink, and we wonder why. Before very long it dawns on us that he needs his bile. This man cannot do without his bile as the others did. We remove the tube to let it close up, but it will not close. The bile still flows out and the patient is still in a serious condition, and we see that patient is going to die, and occasionally one of these patients will die, and I believe he does so because he does not get his bile.

In a few cases we have succeeded in these weakened patients by replacing the tube, catching the bile in a rubber bag and putting it in milk and feeding it to the patient. These patients immediately improve if we can succeed in getting them to take their bile. Some of the patients complained that they did not like the taste of the bile. In some patients I have given it through the stomach tube, and they got along famously. I have sometimes thought it would be advisable to feed many common-duct patients their bile shortly after the operation to prevent their falling into this state of debility from its loss. Perhaps the loss of bile from tubage of the gall-bladder is only partial, and that from direct drainage of the common duct is in many cases complete.

Dr. P. E. Truesdale, Fall River, Mass.:

Permit me to report an observation which would emphasize the importance of classifying all of those patients who have an infection of the pancreas as potentially diabetic:

A woman, aged twenty-eight years, was admitted to our clinic in 1909 with acute hemorrhagic pancreatitis. The operation advocated by Dr. Charles A. Porter, of Boston, was done, laying open the pancreas and draining freely. At the end of three weeks a large portion of the pancreas sloughed out. The patient recovered and she was advised to remain on a carbohydrate-free diet for some time and to continue to exercise reasonable care in the future in regard to the use of carbohydrates in her diet. We followed her for about three years, during which period she remained apparently well. We assumed she had recovered completely, but it was a mistake. About four months ago, after a period of twelve years, she was admitted to the hospital in a state of diabetic coma. She died and the autopsy revealed that there was approximately one-third of the normal bulk of the pancreas left. This patient's life might have been prolonged if we had realized that she was a diabetic and that she ought to have reported frequently for examination.

From her mother it was learned that up to a period of six months

ago she had been careful about her diet, but feeling well she began to eat candy, cake, etc., and this indiscretion was followed by hyperglycemia, diabetic coma and death.

Dr. Kellogg Speed, Chicago:

Last year I reviewed fifty-two cases of carcinoma of the pancreas, and it was my observation that in those cases in which the gall-bladder was drained or removed death followed within six or seven weeks, largely because the patients lost weight and strength and went down rapidly. On the other hand in those cases which were treated by cholecystenterostomy or cholecystgastrostomy, death was delayed for some months—in one case nine months and in several six or seven months. It seems to me very important to know whether we have carcinoma of the pancreas or not, because if we drain or, worse, if we remove the gall-bladder we are taking away the patient's bile, and if we take away his bile death will follow shortly. A diagnosis of carcinoma of the gland is required so that the gall-bladder can be saved to use for a connecting operation, which will return the bile to the individual. We advise in all operations in the gall-tract regions a careful inspection and palpation of the pancreas.

Dr. W. E. Sistrunk, Rochester, Minn. (in closing):

I am very sorry that I cannot answer Dr. Gibbon as to whether pancreatic stones are shown by the *x*-rays. In three of the cases of which I talked today the stones were found accidentally while the pancreas was being examined, and no effort had been made to *x*-ray the pancreas; in one case in which we could have used the *x*-ray we failed to do so.[1]

The chemical analysis of the stones shows them to be composed largely of carbon, phosphorus and magnesium salts. I think stones which contain lime show shadows in *x*-ray plates.

I agree with Dr. Gibbon in believing that when a stone is found in the lower part of the duct a transduodenal operation would probably be best. It is a temptation, however, when you can with one hand push the stone up against the mesentery of the duodenum, not to make a hole through the mesentery and remove it in that way.

Dr. John B. Deaver, Philadelphia (in closing):

Dr. Graham's work is of the utmost value, particularly that on the lymphatics of the liver and the pancreas.

[1] In a personal conversation with Dr. Freeman, of Denver, Col., immediately after this paper was read, he stated that he had recently seen a case in which the *x*-ray plates had shown very clearly multiple stones in the pancreas and that the findings were confirmed at operation.

A recent experience well illustrates the importance of the lymphatic system in abdominal pathology. In a case of acute appendicitis without involvement of the mesocolon the operation was a simple one, consisting of removal of the appendix and closure. Four days later the patient had a chill, followed by fever and sweats twice daily. Upon examination the typhoid bacillus was found in the blood. The patient was transferred to the medical ward. One week later the house doctor decided that the case was not one of typhoid. This is not the first time I have known a case of supposed typhoid turn out to be some other postoperative infection. I suggested opening the abdomen, and the suggestion was agreed to by the patient's family physician and his family. At the second operation I found a marked retroperitoneal lymphangitis, a big liver and spleen. The infection had involved the liver, the gall-bladder and the retroperitoneal lymphatics. Free bile drainage was instituted through the gall-bladder and the common duct and the case went on to satisfactory recovery. This is clinical proof to my mind that Graham is right. My advice is to drain all of these cases as the only logical way of obtaining recovery.

Dr. Evarts A. Graham, St. Louis, Mo. (in closing):

In regard to Dr. Gibbon's statement as to the frequency of cholecystitis and typhoid fever, the point which I wanted to bring out was not at all that typhoid is not frequently a precursor of cholecystitis but rather that cholecystitis seldom occurs during an attack of typhoid. If typhoid cholecystitis were merely a matter of a contact infection of the mucosa we should certainly expect that it would occur in nearly 100 per cent of cases of typhoid fever during the acute stage, because undoubtedly at such a time the bile is teeming with typhoid bacilli, so that multitudes of the organisms are in contact with the mucosa. In 1909 J. Koch[1] thoroughly studied at autopsy a case of cholecystitis which had occurred during the course of an attack of typhoid fever and demonstrated the infecting organisms in the wall of the gall-bladder. He concluded that typhoid cholecystitis is embolic and is not caused by the action of infected bile. He also recognized the presence of infection of the walls of the large bile-ducts. In the same year also Chiarolanza,[2] under Koch's direction, undertook experimental work

[1] Typhusbazillen und Gallenblase, Ztschr, f. Hyg. u. Infectionskr., 1909, lxii, 1.

[2] Experimentelle Untersuchungen über die Beziehungen der Typhusbazillen zu. der Gallenblase und der Gallenwegen, Ztschr. f. Hyg. u. Infectionskr., 1909, lxii, 12.

on typhoid cholecystitis, from which he drew conclusions which, in general, agreed with those of Koch. I should like to mention also that experimentally it is easy to demonstrate that bacteria readily gain access to the bile if sufficient quantities are injected intravenously, especially into the portal vein, without necessarily producing a cholecystitis. This constitutes in reality merely a passage of bacteria through the liver. The point that I have been trying to make is this: In my own opinion, at least, it is necessary to have not merely a passage of organisms through the liver but actually a hepatitis antecedent to the cholecystitis; for the occurrence of a cholecystitis presupposes the presence of virulent organisms, and these would almost certainly induce a hepatitis in their passage through the liver. For similar reasons also I feel that bacteriologic studies of the contents of the gall-bladder do not throw much light on the question of the nature of the organism responsible for the cholecystitis, because organisms may be present in the bile which have nothing to do with the origin of the particular case of cholecystitis in question. Certainly, no one is content to make a bacteriologic diagnosis of appendicitis on cultures made only from the lumen of the appendix; and similarly I do not see the value of cultures made only from the bile in cases of cholecystitis. It is well known, also, of course, that the urine may be teeming with bacteria without the slightest evidence of cystitis.

I do not wish to take the position that all cases of cholecystitis represent a lymphogenous infection. It seems to me, however, that perhaps in the majority of cases the pathogenesis can be best explained in this way. We know that cholecystitis is particularly likely to occur after infections of the portal system. If it were a hematogenous infection of the gall-bladder in most cases then it would seem to me that we might expect its occurrence just as frequently after infections of any part of the body, as, for example, of the hand, but such is not the case. I do not mean to give the impression that Rosenow's work is of no value. On the contrary, I think it is very important. But I do not think that the majority of cases can be so adequately explained, in the light of the conditions that present themselves, on the hematogenous basis as on the lymphogenous basis secondary to an infection of the liver.

I was very much interested in Dr. Archibald's presentation. I think it is a very illuminating piece of work. I cannot quite agree with Dr. Archibald, however, in the assumption that all cases of pancreatitis are essentially the same. It seems to me that the condition of acute hemorrhagic pancreatitis is very different from the chronic interstitial pancreatitis which is so common a sequel of cholecystitis. His experiments deal with the production of acute hemorrhagic pancrea-

titis. I do not believe it is necessary to assume the regurgitation of bile into the pancreas in most cases of chronic pancreatitis, such as occur in association with cholecystitis. It seems to me that they can be better explained on the basis of a spread through the lymphatics to the pancreas.

Dr. Willy Meyer, New York:

One word in reference to the pancreas: Dr. Mayo told us how he had resected a large portion of the pancreas in a case of torsion of the spleen, removing a third, if not half, and there was no trouble whatever. It seems the pancreas stands such resections quite well. In one of my patients with a large sarcoma of the spleen the distal half to one-third of the pancreas was so thoroughly attached that it had to be excised along with the spleen. I tied a stout ligature of chromicized gut around the gland and then amputated it with the actual cautery. The patient made a good recovery.

Regarding prolonged drainage of the gall-bladder in chronic pancreatitis a cholecystitis can, of course, easily occur; but we can wash the gall-bladder with antiseptic fluids the same as we do the urinary bladder and thus overcome the catarrhal affection. I would recommend decidedly, from what I have seen, to drain these cases of chronic pancreatitis to the surface for a long time if chronic inflammatory induration of the head of the pancreas gives the indication.

Regarding the so-called Meltzer-Lyon test there is no doubt that Dr. Lyon has rendered a great service to science and to the investigation of these cases by calling attention to the use of the 25 per cent solution of magnesium sulphate and its effects. I think there is hardly a hospital in this country where every man on the house staff does not take the greatest interest in this class of cases today. However, I had the privilege of being present when Dr. Lyon demonstrated his method and its results before the American Gastroenterological Association at Atlantic City last year. He showed us a flask which contained at least six to eight ounces of black fluid, which he pronounced as having been discharged from the gall-bladder in making the test. Right there and then I was wondering at the tremendous size that this gall-bladder must have had. Further, we all see that *in every instance* the so-called B-bile assumed to come from the gall-bladder is black. Yet if we examine the bile previous to operation with the help of the duodenal tube and the patient in the fasting condition the fluid collected is not always black but is greenish, greenish black, greenish yellow, etc.; sometimes it looks like pea soup. And if we aspirate the gall-bladder during the operation just before excision we find the same variety in color. I believe with Einhorn that the black bile collected

as B-bile, in the Meltzer-Lyon test, does not come from the gall-bladder but mostly from the liver. Therefore, I would say that the Meltzer-Lyon test has with reference to diagnostic purposes no value or but very little value. Therapeutically, however, I believe it has great value, which will likely increase from year to year, thanks to further careful investigation.

Regarding indication, I, of course, do not mean to say that we should extirpate every gall-bladder in patients who complain of pain in the upper abdomen. But the patients in question have gone to the medical man on account of pathologic symptoms. With all diagnostic means at our disposal the stomach, duodenum and pancreas were found to be normal, yet they had symptoms. A duodenal contents examination proved that the gall-bladder was affected. When the abdomen was opened the gall-bladder looked blue and glistening, macroscopically normal, yet with the diagnosis of disease made before operation we knew, from similar instances, that this gall-bladder, though looking not abnormal, was diseased, likely containing pathologic organisms in its wall, and, as we have heard this morning, not in the mucosa but in the deeper strata. According to our present view such a gall-bladder should come out in order to cure the patient.

TYPICAL FIBROMYOMA OF THE ABDOMINAL WALL FOLLOWING HYSTERECTOMY

By GEORGE EMERSON BREWER, M.D.
NEW YORK

The object of this communication is to report and place on record the history of a patient in which a large, typical, fibromyomatous tumor developed in the abdominal wall, in the scar of a previous laparotomy undertaken for the removal of a uterus which was the seat of multiple growths of the same character.

In May, 1911, Mrs. E., aged thirty-one years, sought my advice in regard to treatment of a large tumor of the lower abdomen, which, upon investigation, proved to be a uterine fibroid. As the tumor had been growing rapidly and had already given rise to definite pressure symptoms, an operation for its removal was suggested. Accordingly under ether anesthesia, after the usual preparation, the abdomen was opened in the median line and the uterus, both tubes, and the left ovary were removed. On subsequent examination, the uterus was found to be the seat of several fibromyomata, varying considerably in size, the largest arising from the posterior wall near the fundus, measuring about 50 cm. in circumference. The patient made a satisfactory convalescence, the abdominal wound healing by primary union.

She remained well for nine years. In the late autumn of 1920, however, she began to experience a sense of weight and indefinite pain in the lower abdomen and right side. These symptoms were not constant, but would recur after severe or unusual exertion; and in January, 1921, there was added a slight irritability of the bladder which gradually increased, necessitating frequent micturi-

tion. There was no noticeable interference with nutrition, no loss of strength, and no anemia; but the symptoms gave rise to a certain degree of anxiety and apprehension, which finally led her again to seek my advice.

On physical examination, an oblong, oval tumor was found in the lower abdomen, reaching from the pubis upward and somewhat to the right to a point three inches below the umbilicus. The tumor was moderately movable, was not tender, had a smooth uniform surface and gave to the examining hand the sensation of an ovarian cyst. On bimanual examination these findings were verified, and I was convinced that we had to do with a cyst arising from the remaining right ovary, pressing upon the bladder, and not attached to the cervical stump which was freely movable without transmitting its motion to the tumor. As there was no unfavorable factor revealed by the general physical examination which followed, an operation for the removal of this supposed ovarian cyst was advised. In January, 1921, at the Presbyterian Hospital, under ether anesthesia, an incision was made through the right rectus muscle. As soon as the fibers of this structure were retracted, there was exposed what was thought to be a greatly thickened and highly vascular peritoneum, numerous large veins passing irregularly across the field in various directions and apparently ceasing abruptly as if they penetrated to some deeper structure. Several unsuccessful attempts were made to incise the peritoneum, but the blade of the scalpel opened no cavity, and seemed only to incise a dense solid mass which bled freely. Believing that these conditions only indicated that the cyst was densely adherent to the thickened parietal peritoneum, I divided the tissues carefully, until I exposed a grayish, non-vascular, fibrous membrane, which I thought to be the cyst wall. From this I separated the thickened tissue on either side by blunt dissection, until a sufficient area was exposed to convince me that it was the surface of the tumor. An aspirating needle was then introduced, but without result, other than imparting to the hand the sensation that it passed into a dense fibrous mass.

Still convinced that I was working within the peritoneal cavity,

and that I was dealing with a solid tumor arising from some one of the abdominal viscera, I attempted cautiously still further to expose its surface by separating from it its dense fibrous and vascular envelope. Much to my surprise, as I worked outward from the median line, I was able soon to detect with my finger a distinct line of cleavage, and in a few moments was able easily to enucleate the entire tumor from its bed. After its removal I found that at one point a small opening had been made through the posterior wall of the surrounding membrane into the peritoneal cavity. This opening was enlarged sufficiently to admit my hand, when it was found that the inner layer of the peritoneum was perfectly smooth and free from adhesions with any of the abdominal viscera. This conclusively demonstrated that the growth was an extraperitoneal tumor lying between the peritoneum and the rectus muscles, surrounded by a dense, highly vascular capsule made up of thickened and subperitoneal areolar tissue.

On section the tumor presented the gross appearances of a typical fibromyoma, without any suggestion of malignancy. After accurate hemostasis, the peritoneum was united with catgut, the muscular and aponeurotic structures with chromic gut, and the skin incision closed by silkworm-gut and silk sutures. Her recovery was without accident, and except for the usual post-operative sensations, she has enjoyed the best of health.

On examination the tumor was found to have the following measurements: 16 cm. in length, 13 cm. in breadth, and 9 cm. in the opposite transverse diameter. The appearance of the tumor and the cut surfaces may be seen in Figs. 1 and 2.

The following report on the microscopic examination of the tissue was furnished by Dr. Arthur Purdy Stout, assistant pathologist to the hospital.

"Gross Specimen: Specimen consists of a firm tumor mass measuring 16 x 13 x 9 cm. The surface for the most part is smooth and glistening; it is somewhat nodular. Cut surface shows a dense white tissue which is made up of numerous interlacing bundles of tissue. Tumor weighed two and a quarter pounds.

"Microscopic Examination (Fig. 3): Section shows bundles

and strands of what is apparently fibromuscular tissue. The spaces between these are filled with a rather homogeneous tissue containing elongated nuclei, in places the tumor is exceedingly cellular, and elsewhere there is some evidence of degeneration."

"The Van Giessen stain shows that the tumor is composed almost entirely of fibrous tissue and smooth muscle.

"The Weigert stain shows the absence of elastic fibers."

Diagnosis: Fibromyoma.

From this report it will be seen that the tumor removed from the subperitoneal space in the anterior abdominal wall was a typical fibromyoma, similar in gross and microscopic appearances to the ordinary fibromyomata so commonly found in the uterus.

To account for the development of this tumor in this unusual location, in the writer's opinion, we can consider only four possibilities: (1) That it arose spontaneously from the subperitoneal connective tissue; (2) that it developed from the urachus; (3) that it was of teratomatous origin, and (4) that it was an implantation tumor originating from some minute fragment of the primary tumor left in the abdominal incision at the time of the previous operation.

Before entering into a discussion or critical analysis of these four hypotheses, I may be permitted to state that, as a result of a fairly diligent but by no means exhaustive search of the literature of the subject, in which I have been aided by the advice of Professor W. H. Welch, of Baltimore, Professor James Ewing, of the Cornell Medical School, and Professors Jobling and William C. Clarke, of the School of Medicine of Columbia University, as well as by a number of our best authorities on gynecology and abdominal surgery, I have been unable to find a report of a single case of a typical fibromyoma of the abdominal wall. I have likewise been unable to discover an authentic instance of a tumor of this variety arising from areolar or fatty tissues in any part of the body; and no record has been found of a fibromyoma developing from the urachus, whether patent or obliterated.

These facts, and the recognized impossibility of leiomyomatous tumors developing from tissues containing no smooth or unstriated muscular fibers, would enable us at once to exclude the first hypo-

thesis, that such a neoplasm could spontaneously develop in the subperitoneal areolar and fatty layer of the abdominal wall.

FIG. 1.—Photograph of gross specimen—removed from the abdominal wall.

In regard to the urachal origin of our growth, one had to be somewhat more guarded, as the urachus is known to contain a

definite layer of unstriated muscle; and even after birth these muscular fibers generally can be demonstrated in the fibrous cord

FIG. 2.—Cut surface of tumor.

representing the obliterated canal. The absence, however, of any recorded example of a fibromyoma arising from this structure, and

the absence of any evidence in this case of an attachment to anything resembling the urachal cord, would render our second possibility highly improbable.

In regard to the third, or teratomatous origin of the tumor, it must be admitted that the median line of the abdomen is a well-recognized habitat for these minute islands of embryonal tissue and numerous examples of teratomatous growths from these rests have been recorded.

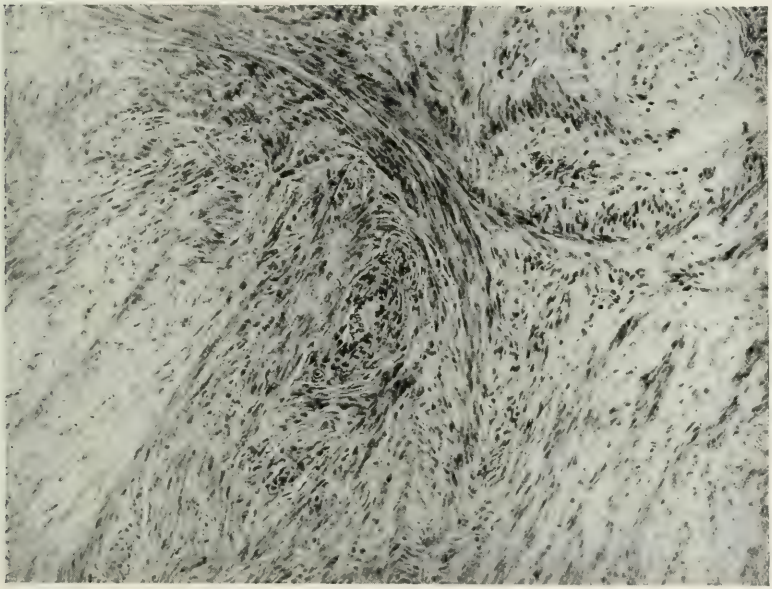

FIG. 3.—Microscopic appearance of tumor.

In all such cases, however, that have been carefully examined, although one type of tissue may preponderate and make up the great mass of the tumor, a painstaking search will always reveal the presence of other types of tissue in some part of the neoplasm. The absence in this instance of any evidence of the presence of other types of embryonal tissue would lead one logically to exclude this explanation of its origin.

In regard to the fourth hypothesis, that it arose from the implantation, in the line of incision, of a minute fragment of the uterine tumor removed ten years before, the writer is of the opinion that this is by far the most probable and reasonable explanation of its occurrence. This opinion is based upon the following facts: (1) The coincidence of the development of this tumor in an individual who had previously harbored an exactly similar growth in a neighboring structure; (2) that in its removal the tumor-bearing organ with its divided and exposed tissues had been for some time in actual physical contact with the divided and exposed tissues of the abdominal wall, in the exact location in which the secondary growth subsequently developed; (3) the well-known and generally recognized fact, that certain cutaneous and mucous membrane tumors which are in close or frequent contact with neighboring tissues, not infrequently give rise to similar growths in the tissues thus exposed; (4) that modern experimental surgery has furnished innumerable examples of successful grafts of tumor tissue as well as normal glandular structures, in both animal and man, and (5) that surgical literature records not a few examples of both benign and malignant tumors arising in the immediate neighborhood of incisions made for the removal of histologically similar growths; as well as tumors developing from the accidental implantation of aberrant fragments of normal tissues or cells into a new and histologically different environment.

As examples of the first may be mentioned the not infrequent development in an abdominal scar, of a malignant growth after operation for the removal of a similar neoplasm of one of the viscera, and the occasional recurrence of ovarian cystomata or adenomyomata after similar procedures.

In a recent article by Cullen, published in *Archives of Surgery*, September, 1920, entitled "The Distribution of Adenomyomas Containing Uterine Mucosa," three such cases are reported involving the abdominal wall, one in which the uterus was ruptured during a curettement for abortion with immediate laparotomy for surgical repair of the uterine wound, and two others following operations on the uterus or adnexa.

The writer may be permitted to state that his opinion as to the implantation origin of the tumor reported in this communication was greatly strengthened by a personal letter received from Professor Welch, in which the following view was expressed:

"I find no difficulty in accepting your suggestion of the origin by implantation of the fibromyoma of the abdominal wall, and this seems to me the most probable explanation. What speaks for the implantation origin in your case is (1) the location precisely in the scar of incision, and (2) the obvious opportunity for implantation at the time of the previous operation. Much more complicated ovarian cystomata, for example, may thus arise by implantation in abdominal scars.

"I know of no case identical with yours, and this exceptional occurrence indicates that the conditions for a successful graft by implantation at operation on uterine myomata must be very rarely met, but myomata may develop from cells transplanted from an original tumor, and there is no inherent improbability in my opinion in assuming the similar origin by implantation in your case. I see no reason to think of a possible teratomatous origin."

Although the writer from his limited search of the literature would not presume to claim that the finding of this tumor in this unusual locality was a unique occurrence, still, from the evidence reported, the condition must be one of extreme rarity, and for that reason he has thought it worthy of presentation to this Association.

SURGICAL ASPECT OF TUMOR OF THE BRAIN

By MILES F. PORTER, M.D.
FORT WAYNE, IND.

Both the morbidity and the mortality caused by all tumors of the brain, all tumors of the meninges and all tumors of the inner aspect of the skull result largely from increased intracranial tension, local, general or both. The word tumor as here used is intended to apply also to infectious granulomata. These pressure phenomena are the result of an increase in the intracranial content—they are mechanical in origin. In 100 cases studied in the service of Drs. Halsted, Heuer and Dandy[1] they found that in 57 evidence of the increased intracranial pressure was manifested in the *x*-ray pictures by a separation of sutures. Mechanical defects are best remedied by mechanical measures. These statements, in the present state of knowledge, must, it seems to the writer, be accepted as true. They require qualification and exemplification, but in a general way they present a true picture. Logically, therefore, two facts seem to follow: (1) That all tumors occupying the cranial cavity should be taken into consideration in a discussion of brain tumors, as is done by Hertzler[2] and other writers. (2) That the subject of brain tumor is a surgical rather than a medical subject.

Last summer, in Paris, the writer was present at a clinic conducted by a surgeon of decidedly excellent repute. One of the cases was listed as "brain tumor." The operator proceeded to remove a large part of the frontal bone and, very much to his surprise, when the segment of bone came away, the tumor came with it. Technically, it was a tumor of the skull; practically, it

was a tumor of the brain. This is not intended as an adverse criticism, for the operation was well conceived and splendidly executed, but the incident is related in support of the contention that all tumors occupying the cranial cavity should be considered in discussing the general question as to whether tumors of the brain should be considered as primarily surgical rather than medical. It would seem obvious that if the majority of patients with brain tumor can be relieved or cured by surgery only, then these patients should be considered surgical subjects until the contrary is proven.

As late as 1902 Ransohoff,[3] in a paper read before the Surgical Section of the American Medical Association, said: "Only 6 per cent of all cases are suitable for operation." Bergmann, as quoted by Hoppe,[4] came to a like conclusion as the result of a study of 673 cases taken from various sources.

The writer takes it that these authors mean to convey the idea that only about 6 per cent of tumors of the brain *per se* can be removed surgically. Certainly more than 6 per cent of patients with tumors of the brain proper can be relieved surgically and a still larger per cent can be relieved by surgery if tumors of the meninges and inner table of the skull are included. Ransohoff puts the three-year recoveries from operation in brain tubercle at over 25 per cent. As emphasized by Cushing[5] it is oftentimes impossible to differentiate between true tumor and what he calls pseudotumor cerebri. He further quotes and supports the statement of Tooth that even among the fatal cases are "many positive surgical successes, some almost brilliant considering the circumstances, and followed by relief of pain, improvement in sight and mental state and prolongation of life." Cushing operated upon 350 out of a series of 500. Headache is an almost constant symptom of tumor of the brain, and according to Paton, as quoted by Rawling,[6] either optic neuritis or atrophy occurred in 81.2 per cent of 252 cases. Surgery, and surgery only, can relieve either or both of these symptoms in a large percentage of cases. Beriel[7] operated 8 out of 16 cases of glioma and advises calling the surgeon as soon as the diagnosis

is made, but insists that no attempt be made to remove the tumor because simple decompression gives better results. The position taken by White,[8] Seguin,[9] v. Bergmann[10] and Koplik[11], that gummata, non-localizable tumors, non-encapsulated tumor, tubercular tumor and tumors of any sort in comatose patients are non-operable, is no longer tenable. As long ago as 1892 Czerny[12] expressed the opinion that pathologico-anatomical results were not authoritative in brain tumor. Wyeth,[13] prior to 1894, operated two patients in coma due to brain tumor. Both patients became conscious and one was able to attend to business for seven months and to execute a will which stood. In neither case was the tumor removable. It is generally conceded now by surgeons of experience that decompression may be expected to alleviate the distressing symptoms of brain tumor regardless of its location or nature; but it is a humiliating fact proven by an extensive search of the literature that, notwithstanding the advice of Cushing,[14] Sachs,[15] Sharpe[16] and others, a large number of patients are allowed to suffer an unconscionable time, amounting in the aggregate to decades, for the want of timely, intelligent, surgical interference. Early operation not only relieves the symptoms but tends to prevent the secondary changes which occur in the brain as a result of the tumor—the hyperplasia of Spiller.[17] To allow a patient to go blind or even to allow him to suffer over a long period in the hope of making a complete diagnosis, or in the hope of relief through antisyphilitic treatment, is almost a crime. The time which is considered reasonable during which antisyphilitic treatment may be tried is rapidly growing shorter. In 1893, C. H. Mayo[18] said, "Tumors diagnosed as gumma should be treated medically for three months." Horsley,[19] in 1910, put the time limit for antiluetic treatment at six or eight weeks at the most; while Rogers,[20] in 1911, says "from three to six weeks is long enough to test the utility of antisyphilitic remedies," and Sachs,[21] in 1920, puts the limit for palliative treatment at four weeks.

Especially in children is it necessary to guard against losing valuable time in the hope of giving relief by antisyphilitic medi-

cation. Rivista[22] found only one gumma in 140 cases of brain tumor in children, and in this case the child died after nine months of antisyphilitic treatment, which treatment did not seem to modify the gumma.

Frazier, in a letter to the writer, says, speaking of gummata: "In the first place, it is unimportant in the majority of instances to make a distinction, and in the second place, after you think you have made it, you probably find you have made a mistake, and an operation is often necessary to confirm diagnosis and to deal adequately with the lesion. To make a diagnosis of gumma does not exclude it from the possibility of a surgical lesion." It is well to recall here the observation of MacEwen, made at the International Medical Congress in 1909, to the effect that avascular gummata would respond to antiluetic treatment only after pressure had been relieved.

It is well to recall here also the fact that decided remission in symptoms does not exclude brain tumor, nor does it indicate necessarily that the treatment used was the cause of the remission. Abercrombie, in his work on *Diseases of the Brain*, published in 1833, relates the case of a patient who became blind rather suddenly, whose sight was restored by an emetic, but lost again, and permanently, within an hour. Rudolf and MacKenzie (*Amer. Jour. Med. Sci.*, cxxxviii, 733–1909) report a very interesting case illustrating the extraordinary remissions that sometimes occur in patients with brain tumor.

Ballance states the case about right, in my opinion, when he says: "Decompression should be done before symptoms have become more than barely sufficient to enable a probable diagnosis of brain tumor to be made; one should never wait for pressure symptoms in the eye to develop." Cushing, in a recent letter to the writer, said: "I have seen patients with syphilomas become blind during treatment for them. I think a syphiloma is better treated like any other tumor."

Given a diagnosis of brain tumor the patient's interests are best served by an immediate operation; waiting for a complete diagnosis is dangerous. If for any reason delay in a given case

is desirable, the time of the delay should not be arbitrarily limited on the authority of any one, but rather the case should be carefully watched, especially the eye grounds, and the operation done before irreparable damage has occurred. Then, too, the suffering which some of these patients endure may be, in and of itself, a sufficient warrant for a decompression operation. Personally, I am in favor of the suboccipital precedure in subtentorial tumors; however, as Cushing[23] suggests, it is questionable whether a decompression here is any more effectual than one placed elsewhere. For all other than subtentorial tumors Cushing's subtemporal operation is the one of choice, unless one desires to-make some exploration in the hope of locating the tumor at the same time, in which case the operation may be planned along the lines advised by Hudson,[24] or the combined subtemporal decompression and exploratory operation described by Cushing.[25]

In 1902, and again in 1903, the writer[26] called attention to the use of the exploring needle as an aid in locating tumors, abscesses and cysts of the brain through the change in resistance transmitted to the surgeon's hand as the needle progresses. Since this time I have frequently referred to this diagnostic aid in discussing the subject, but for some reason it has never come into general use. The hollow needle is quite frequently used in cysts and abscesses, but one rarely sees a report of the use of the solid needle in the way suggested. In a recent letter Dr. Cushing says that "needling for brain tumor is employed by all so-called neuro-surgeons." The evidence contradicts this statement, in so far as concerns the use of the solid needle in the way and for the purpose here advocated. *Die Hirnpunction* of Neisser and Pollack and Krause[27] contemplates the use of a hollow, sharp needle, with its attendant dangers, for the express purpose of determining the presence or absence of fluid and for tapping the ventricles. The value of the needle in locating tumors and abscesses I have demonstrated, and Elsberg[28] also has discovered tumors in this way, which he says "would otherwise have been missed." However, the reports of cases make it plain that many exploratory operations for brain tumor result in failure because

this simple, safe and valuable expedient is not generally known and used. In 1912 Willy Meyer[29] said, in speaking of an exploratory operation for supposed cerebellar tumor, "The entire cerebellum was exposed and carefully explored with negative results. The patient died, and at the autopsy a large cyst was found in the left hemisphere, involving temporal and occipital lobe. Were it not that the sometimes dangerous consequences of aspirating the brain made surgeons fear the multiple use of the needle in intracranial operations, the cyst in this case might have been found and evacuated." I venture the assertion that this cyst might have been discovered by the use of the solid needle. Cushing,[30] in his article on a combined exploratory and decompressing operation, cites two instances of tumor "too far below the cortex to be identified." It makes little or no difference how far below the cortex the tumor is situated so far as concerns its detection by the use of the needle. The detection rests upon the difference in resistance offered to the progress of the needle by the normal as compared with the abnormal tissue. Spiller[31] says: "Deep probings where the tumor is not upon the surface are rarely of value and do harm." With the use of the solid needle the danger of "aspirating" the brain is eliminated, and from experience and study I am convinced that the precedure is relatively without danger and of great diagnostic value. The needle used should be long, slender, round and rather dull. Elsberg has used a slightly roughened needle, and says by this means one is often able to recognize slight differences of resistance in the brain. I have not used a roughened needle in actual work, but think the suggestion valuable and shall act on it in the future. A fine trocar and cannula, such as is used for intraspinous work, dulled slightly at the point and slightly roughened for about an inch at the distal end, makes an ideal instrument for exploring the brain. Coupled with the solid needle, one has also the canula in case he wants to tap a cyst or an abscess.

The writer has experimented with such a needle by imbedding in a fresh brain pieces of liver and then locating them by the sense of touch as imparted through the needle. He has also

demonstrated the possibility of locating vessels and ducts in liver substance with the needle, and he ventures to suggest to neurologic surgeons, who have not had experience with the solid needle for the purpose of locating tumors of the brain, that if they will repeat these experiments they will be convinced of the value of the method.

With two- or three-stage operations I have no experience and, like Cushing, I do not look upon them with favor. However, it must be conceded that in some instances they become a necessity because of shock or hemorrhage or both, and in other instances advisable where one has failed to locate the tumor at the first operation, in the hope that relief of pressure may make localization possible later on. The necessity for a two-stage operation for simple decompression must be very rare. It should be remembered, too, that some surgeons of large experience, like Von Eiselsberg,[32] prefer the two-stage operation for the removal of tumors, on the ground that it is safer for the patient.

Drainage in this work should be looked upon as an evil which in some cases becomes a necessity. When employed it should be removed in forty-eight or seventy-two hours. Permanent drainage would prolong life and minimize the deformity in those cases in which it is impossible to remove the pathology. However, I know of no reliable and safe way of accomplishing this. G. A. Cathey[33] makes a tube of the dura and says that this "establishes permanent drainage," but I have not seen his original article and am unable to say upon what evidence his statement is based. We do know, however, that, generally speaking, fascial tube drains are disappointing. Halsted and Vaughan[34] say: "*X*-rays, radium and fulguration practically do not come into consideration in the treatment of intracranial tumors." Later reports are more encouraging, however. S. Nordentoft[35] has a record of 20 cases of brain tumor treated by roentgen exposures with encouraging results. The favorable results in his "first series of 8 cases have continued to date; there has been no recurrence of the growth or return of symptoms." Muskens[36] reports gratifying results from roentgen-ray treatment in 2 cases

of cerebellopontile tumor. Frazier[37] reports 3 cases treated with radium, in which the "growth of the tumor, in the light of the clinical evidence, has been arrested at least, the tumor possibly destroyed in periods of observation covering a maximum of eight years." In one of Frazier's cases the radium was buried in the tumor, in the pontine angle, in one (also cerebellar) the tumor was not exposed and four applications of radium were made following suboccipital craniotomy. In his third case the treatment consisted of combined use of *x*-ray and radium, together with pituitary and thyroid feeding following a sella decompression. Williamson, Brown and Butler,[38] as a result of experiments with radium upon dogs for the purpose of determining the safe dosage of radium, say that "the experiments give assurance that in brain tumors which respond readily to radium, little or no damage will be inflicted upon the brain tissue surrounding the tumor if the radium is implanted in the growth. The dosage can be regulated so as to be destructive only to the periphery." Unfortunately, Frazier found no case of glioma which seemed to be favorably affected by radium. On the whole, however, the later reports as to the effect of radium and *x*-rays on brain tumors are very encouraging.

Conclusions. 1. The term "brain tumor" should include all growths of whatever origin or nature, either in or on the brain.

2. Brain tumor is essentially a surgical malady.

3. Surgical intervention should follow promptly the diagnosis of brain tumor. Postponement of operation for the purpose of locating the tumor or in the hope of getting relief through antiluetic treatment or for the purpose of determining its character is seldom warranted. Better a comfortable, contented patient without a complete diagnosis than a dead patient or a blind one with one.

4. Exploration of the brain with a solid needle is a valuable aid in the localization of tumors and is free from danger if properly done.

5. *X*-ray or radium therapy should be used in all inoperable cases in which surgical removal is impossible, and after removal of malignant tumors.

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THE CANCER PROBLEM

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The cancer problem has been the subject of investigation for more than two thousand years and still remains a question of superlative importance which is engaging the attention of governments, societies, commissions and the greatest endeavors of the medical profession working together and singly in the hope of a solution.

Vast sums of money have been spent in the collection of facts and figures; in the furthering of research work; in the founding, equipping, staffing and maintaining of special hospitals and clinics where cases may be treated and studied; in the education of the people and workers on cancer. The result is that at present no less than fifteen fully equipped cancer hospitals are treating patients, twenty research laboratories are working continually, to say nothing of the innumerable individual laboratories in America and Europe; each university is contributing its quota; every pathological laboratory is making known facts and findings which assist in throwing light on the problem.

All these efforts have succeeded in collecting about the problem such an enormous mass of facts that, while a very great deal has been made clear, the problem has been more or less split into numerous entities for which there does not appear to be a common cause.

The advances have been mainly in the line of classification and in the establishing of the minute structure and behavior of the different forms; and these have occurred since the advent of

the microscope. Prior to this the imagination was given full play in the supposed causation, the diagnosis, and the treatment of the disease. The name given to the disease, which was thought to resemble a crab, illustrates the state of mind of the early investigators. Even as late as the seventeenth century, cancer was thought to be caused by a spiritual being, the Archæus, resident in the stomach and spleen.

The powers of fantasy displayed by the ancients in the formulation of their theories of causation were only equaled by their ability to mystify the methods of treatment. The cancer patient was full partner in all these perpetrations, welcoming anything new and placing favor in the most weird. If he resided in England he was subjected to such mixtures as dog's fennel, crow's feet, sulphur and arsenic; and if he survived these he was qualified to consult the witch doctor, who initiated him into the black art and treated him to some witch's broth. If faith has ever cured cancer, surely it missed a grand opportunity in these manipulations of the occult science. If the sufferer lived in France he grew accustomed, on survival, to all the various ingredients of green frogs. In Germany he drank copiously of concoctions of suckling puppies; while in Africa he could be treated to nothing better than tortoise liver and cancer bush.

How all these treatments originated would occupy too much time in the telling, and would be as difficult to explain as it is impossible to know why some patients today forego an orthodox treatment and feed raw beefsteaks to open malignant tumors, which, they are observed to say, devour the steaks with avidity.

While mysticism seemed to reign in the sphere of treatment, we must observe that as early as A.D. 180 Leonides, of Alexandria, practiced excision of malignant growths by cutting through healthy tissues with the knife and cautery. That after nearly two thousand years this method should find most favor in the medical profession testifies to the integrity of some of the early observers.

While the search and research for the cause of cancer goes forward the profession will carry out the orthodox treatment in

every case of cancer which presents. We should let the waiting world know that until the cause is known and a specific treatment discovered there is one cure for cancer and that cure depends upon early recognition, allowing of early complete excision. And let them know that the *x*-ray and radium come after, in making doubly certain of no recurrence. In taking such a stand the profession would serve a very useful purpose in educating the victim of cancer and his friends, and would materially lower the mortality rate, which today—with more cases diagnosed—has been made to appear on the increase.

There is little excuse for fatal termination in those cases of cancer which have their origin on the surface of the body, for with our present knowledge of the onset, the conduct and structure of cancer an early diagnosis should not be a difficult task. A confusion between a benign and a malignant tumor should be met by the same method of complete early excision. The hidden cancer which comes to light only when invading the body functions, or as secondary growths, is the form which cries out most loudly for a knowledge of the causation upon which adequate treatment may be based. It is in these forms that surgery, at times, can only prolong life; occasionally a judgment of inoperability must be made and the patient placed in the hopeless category with a certain length of time to live. What should be the attitude of the medical profession toward such a patient? His only hope lies in a specific treatment, which is not available. To meet this situation I would suggest that efforts be made for the establishment of a hospital in every large center where these patients would receive the most appropriate treatment and at the same time be subject to investigation.

The Criteria of Therapeutic Effectiveness in Human Cancer

In determining the effects of any given mode of treatment on a tumor the majority of observers lay stress on—

1. The circulatory changes in the tumor.

2. The relief of pain.

3. The restoration of secondarily impaired function.

The foregoing points may be useful in a purely inflammatory process, but are of little or no value in judging the effects of therapeutic methods in malignant disease.

The observation of the size of the tumor itself is the sole criterion on which to judge the effect of therapeutic measures. But even this is unreliable. The fallacy of relying upon a diminution in size is shown by the fact that the tumor may advance at one point and recede at another—that cicatrization and contracture may reduce its size and give a false impression of partial recovery. Finally, stress has been laid on the prevention of metastasis or delay of recurrence. This statement also is misleading, for incomplete removal may be followed by apparent recovery for a long time without local recurrence.

In estimating the value of any new form of treatment the following facts must be borne in mind:

1. Good results at times are obtained in cases supposed to be cancer, but in which the nature of the condition has not been established on incontrovertible evidence. In such cases, the usual conclusion of the medical attendant is, *a priori*, that his diagnosis was wrong, rather than that a cure of cancer had been effected.

2. Sufficient time must elapse to judge the ultimate results. Temporary improvement frequently occurs after the removal of mechanical obstruction by operation or by sloughing of a cancerous growth.

3. Retrogression or even spontaneous disappearance may occur in cases of cancer, so that it is not uncommon to get considerable temporary symptomatic improvement apart from any treatment.

I should like to report a case which came under my observation, ten years ago, of a young woman, aged twenty-one, with a very large inoperable sarcoma of the neck. She had radium and *x*-ray treatment without effect. When she appeared to have but a few months to live her father took her to England. While walking

on the deck of the ship during a storm she fell with great force, giving her neck a severe wrench. The slight increase in swelling which immediately followed was soon succeeded by a reduction in size, until at the end of a few months the tumor was entirely gone. It is now ten years since its spontaneous disappearance, and there is no recurrence.

4. Psychic influences are of great importance. This aspect of the case has been well emphasized by Weil, as follows: "Is it, indeed, very remarkable that a patient who has been consigned to death as a victim of a hopeless malady should regain his spirits and his appetite when he is again confronted with the hope of a cure and of the eradication of his disease? It is a phenomenon well-known to every student of the disease that a large proportion of cases respond in just this manner to any treatment which is offered them. Osler has described a case of cancer of the stomach in which the mere visit of a consultant of sanguine temperament, though poor judgment, whose assurance to the patient that there was no possibility of cancer, resulted in a disappearance of all the symptoms and a gain of eighteen pounds in weight. It is this psychic influence which has occasionally deluded the honest student of cancer cure and which has also so generously played into the hands of the dishonest."

The following are some of the therapeutic agents which have been advocated from time to time:

1. *Cancer Antisera.* Although a vast amount of labor has been expended on cytolytic sera it is now admitted to have failed in producing a therapeutic agent for cancer. The records of attempts to influence the growth of animal tumors by means of heterogenetic antisera are now quite numerous. In no instance has it been possible to determine that heterologous antisera prepared against tumors have a marked cytolytic effect *in vitro* or a curative action *in vivo.*

2. *Vaccination.* Experimental cancer research, thus far, gives practically no hope that human tumor can be affected by means of vaccination as a therapeutic procedure, (*a*) either by the injection of the patient's own fluids or (*b*) of ground-up tumors.

3. *Other Biological Methods. Doyen's Vaccine.* (*a*) Doyen, described a small coccus, which he named Micrococcus neoformans, as the cause of cancer, and from which a vaccine was prepared which he claimed gave 20 per cent. of cures. Under Sir Almroth Wright's direction the method was carefully tested out in the Middlesex Hospital and found to be valueless.

(*b*) *Dr. Glover's Serum.* Dr. Glover claims: That he has isolated and cultured an intracellular organism which is found in every type of cancer. That he has produced a serum by injecting "a roan horse." That this serum renders animals immune to inoculation by cancer cells and will produce improvement or cure in cases of human cancer.

The claims of Glover are very similar to those of Doyen, whose vaccine as stated above was found to be valueless.

I was a member of the committee of the Academy of Medicine, Toronto, on the Glover serum. Dr. Glover refused to permit the committee to visit his laboratories to examine his cultures and experimental material, and it therefore had no evidence to substantiate Dr. Glover's claims on the experimental aspect of the question. From the examination of cases and all available data the committee found no evidence that a cure had been produced by the serum in any case definitely established as cancer.

A number of cases treated by Dr. Glover's serum have been admitted into the various hospitals in Toronto, where portions of growths were removed and sections examined. These sections show no change in the cancer cells and no arrest of the growth. This is in striking contrast to the findings after the use of *x*-ray and radium, where the cells become vacuolated, etc. In addition to the above, we have seen a number of cases of cancer going on to a fatal termination after the use of Dr. Glover's serum since the Academy's report, confirming its findings and justifying the conclusion that Dr. Glover's serum is quite without value as a cure for cancer.

(*c*) *Cancroin* is the name given by Adamkievicz to a 25 per cent watery solution of neurin, which is supposed to be identical with the toxic base of cancer. It was shown to be useless.

(*d*) *Antimeristem*, the name given by Schmidt to a culture of the supposed microbial agent of malignant tumors. It also appears to be quite valueless.

(*e*) *Coley's Fluid: The toxins of erysipelas and Bacillus prodigiosus.* Coley claims 10 per cent of cures. If you balance against this the marked constitutional disturbance produced by the toxins, often with severe pain and (evident) shortening of life of the 90 per cent upon whose growth it produces no effect, it can scarcely be regarded as a remedy that can be recommended even in inoperable cases.

X-ray. Cells exposed to the action of the *x*-rays become vacuolated, do not take the usual stains and the cell wall disappears. In a small superficial growth, such as rodent ulcer, the cells are penetrated by small round cells and leukocytes, which rapidly organize. Thus the growth slowly atrophies and disappears and is replaced by connective tissue. Around the normal edges an inflammatory reaction occurs. It is worthy of note that cancer cells of different types may be radiosensitive in varying degrees.

The problem of *x*-ray therapy of cancer resolves itself into the administration of the correct dose in superficial cancer.

The roentgenologists are suggesting that a number of treatments by *x*-ray be given in cases of cancer *before* as well as *after* operation. I personally cannot see any advantage in delaying an operation for cancer for the purpose of preliminary radiation. After the growth is removed radiation is always employed and seems of value. Roentgenologists are also claiming to be able to cure superficial cancer, such as rodent ulcer and epithelioma of the lip. I take issue with them in recommending this as a substitute for operation. Why should treatments with *x*-ray or radium be continued for weeks or months when a ten-minute operation will remove this superficial growth. In a superficial ulcer it is quite possible to get healing by the use of the *x*-ray and a conclusion be reached that the disease is cured, while at the same time some deep cells have not been reached and later manifest themselves as metastasis in the deep glands. Further,

while the treatment of the superficial ulcer is going on, metastasis may be developing in the glands.

In deep cancer, *x*-ray may be used (1) as a palliative measure in inoperable cases; (2) as a prophylactic postoperative measure. Some roentgenologists are recommending and using a ray of much greater intensity and penetration. I fear, however, that this is not without risk of severe burning or sloughing and have seen cases where this has occurred.

Radium. Practically all that has been said in regard to *x*-ray applies with equal force to radium. Radium seems to have a special place in carcinoma of the cervix uteri. Our position in regard to the use of *x*-ray and radium should be made very clear. I take it that these methods are used only to supplement surgery and not to replace it.

Surgery still offers the best means of relief for cancer, and when the condition is recognized early enough a cure may be effected in a considerable proportion of cases.

A word should be said in commendation of the excellent work done by the Cancer Committee of the American Medical Association in helping to educate the public upon the paramount importance of seeking early advice in regard to any unusual lump or swelling.

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AN ANALYSIS OF ONE HUNDRED AND TWO CASES OF TUMOR OF THE BREAST

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The *American Text-book of Surgery*, published in 1903, states that "about 83 per cent of all mammary tumors are carcinomata, while the remaining 17 per cent are sarcomata, adenomata and cysts." In 1911 my son, Dr. Nathan Winslow,[1] and I tabulated a series of 100 cases of lesions of the breast, occurring in the University Hospital, Baltimore, with the following result: 63 were carcinomata, 3 sarcomata, 20 fibro-adenomata, 1 peri-canalicular myxoma, 5 cystic fibro-adenomata, 2 galactoceles, 3 tubercular mastitis and 3 abscesses. Of these 99 were females and 1 a man, with a fibro-adenoma; 88 were of the white race and 12 of the colored.

The question of the incidence of carcinoma in comparison with all other tumors of the breast seems still to be unsettled; thus Finney[2] places the proportion as 80 per cent cancer; A. P. C. Ashhurst[3] as 70 per cent malignant and Da Costa[4] says malignant tumors of the mammary gland are ten times more common than innocent tumors. Dr. Miles F. Porter,[5] writing in 1919, says that of 77 cases of neoplasms of the breast observed by him personally, 49, or 63.63 per cent, were malignant and 28, or 36.37 per cent, were benign. On the other hand, Bloodgood[6] gives the percentage of benign to malignant lesions of the breast, as observed at the Johns Hopkins Clinic, as gradually rising from 32 per cent, in the first decade after the opening of the hospital, to 59 per cent in the three years immediately preceding his report,

and this he attributes to the education of the profession and the public, through which patients are referred to the clinic at an earlier date than formerly.

As this statement is at variance with the usually accepted opinion, we have gone over the records of the University Hospital for the three years 1917, 1918 and 1919, to ascertain if there has been any material change in the statistics of breast lesions as they have been observed in this particular institution since the publication of our report in 1911. During these three years 102 patients were admitted to the hospital with lesions of the breast of various kinds. Of these, 61 were carcinomata, 3 sarcomata, 18 benign solid growths and 20 benign cystic growths. 99 females and 3 males. Of the women, 90 were white and 9 colored. The men were white and had non-malignant neoplasms. 88 were married, 10 single, and in 4 the social condition is not recorded. The left breast was involved in 56 cases, the right in 44; not mentioned, 2. Both breasts were involved in 1 case, the lesions being benign; 92 cases were subjected to operation, of whom 3 died; 1 of pneumonia, 1 myocarditis and 1 acute dilatation of the heart; 6 cases were inoperable and 3 refused operation; 59.8 per cent were carcinomata, 2.94 per cent sarcomata and 37.26 per cent benign. The length of time that elapsed between the discovery of a tumor by the patient and the application to the surgeon for relief varied from one week in 2 cases to forty-nine years in 1; the largest number of cases coming under surgical care at the end of one year. The ages of the patients varied from nine years, in the case of a negro girl who was operated on under a diagnosis of fibro-adenoma, to seventy-two years in two cases of carcinoma.

Carcinoma. The cases of carcinoma were 61 in number, or 59.8 per cent. The largest number of cases came under observation in the fifth decade of life; 6 occurred before thirty-five years of age and 2 between twenty and thirty years. The youngest was twenty-three years old, and was operated on under the diagnosis of fibro-adenoma, but the pathological report was "cystic papillo-adenoma, undergoing early malignancy." Among

the cases tabulated in our previous report was that of a girl of seventeen, with a fibro-adenoma undergoing malignancy; 2 women aged seventy-two years were operated on, one dying in about three weeks with myocarditis and the other died a year later with a recurrence.

The carcinomata were subdivided into simple carcinoma 28, scirrhus 18, scirrhus with colloid degeneration 1, scirrhus with hyaline degeneration 2, medullary 1, adenocarcinoma 3, cyst-adenocarcinoma 1, cystic papillo-adenocarcinoma 1, cystic carcinoma 1, suppurating carcinoma 1. 57 of the women were married, 2 single and 2 not stated. The right breast was involved in 27, the left in 33 cases.

The tumor was located in the upper and outer quadrant in 22, upper and inner 4, lower and outer 4, lower and inner 1, central 3, general 8, the rest not recorded. For some reason unknown to me the cancerous growth is generally located in the upper and outer quadrant. In my personal experience cancers situated in the upper and inner quadrant are much more unfavorable as regards permanent cure than those located on the axillary border.

In January, 1917, a lady, aged forty-eight years, consulted me for a lump in the upper and inner portion of the left breast. The growth had been in existence six years and had increased rapidly during the preceding six months. There were marked axillary metastases. A radical operation was done and she made a speedy recovery. One year later she returned with a considerable mass in the sternum, opposite the second and third costal cartilages. I excised a considerable portion of the sternum, with the overlying tissues, leaving a large square hole leading into the anterior mediastinum. The internal mammary artery was cut and bled freely before it could be ligated and the left pleural cavity was opened and air sucked in. She developed an empyema, which was drained posteriorly, and she recovered, but died in November, 1919, of general carcinomatosis.

In October, 1917, a colored woman, aged forty-two years, appeared with a fairly movable tumor, as large as a hickory nut,

that had been noticed about six weeks. This was also located in the upper and inner periphery of the left breast and was supposed to be an adenofibroma until a frozen section revealed it to be a carcinoma. The breast, muscles and axillary glands were removed. In the summer of 1919, she returned with a metastasis, very similar to the one mentioned above, in the sternum. She elected to try radium, under the application of which the swelling subsided somewhat. She is still alive but is failing rapidly.

The growth was stated to have been adherent to the skin or muscles in 40 cases and in only 10 was it recorded that it was not attached to either.

The axillary glands were recorded as not palpable in 3 cases and in 2 the supracalvicular glands were to be felt. Duration of the tumor: three weeks 1, seven weeks 1, two months 3, three months 3, four months 3, five months 2, six months 4, seven months 1, eight months 1, nine months 1, ten months 1, one year 5, one and a half years 1, two years 3, six years 3, ten years 1, twelve years 1, twenty years 1, twenty-two years 1, forty-nine years 1.

Many more married than single women have cancer of the breast, but this depends more on fecundity than on the social condition. Trauma also seems to play only a small part in the production of cancer of the breast, and in only 4 cases was there any history of an injury.

Pain, also, is not a frequent concomitant of mammary cancer, and in only a few of our records is it stated that pain or tenderness was present.

One woman had had bleeding from the right nipple intermittently for one and a half years. The nipple is stated to have been retracted in 12 cases, not retracted in 11, and stiff in 1.

There were 6 cases of carcinoma of the breast in negro women. It is a mistaken idea that negroes are to any large extent immune to cancer.

Metastasis to the skeleton is only occasionally observed and the only bone involved in this series, except the two in which the sternum was the seat of recurrence, was the ilium. When

remote metastases occur the usual locations are the spinal column, the femur or the humerus.

Sarcomata. There were 3 cases of sarcoma, or 2.94 per cent. They were all white women and married. Their ages were twenty, fifty-four and sixty-three years. One had been in existence two years and one only two months. In all the left breast was affected.

The pathologic classification was sarcoma 1, myxosarcoma 1, fibromyxosarcoma 1. No metastases were mentioned in any of these cases. Radical removal was done in all, except that in one case the pectoral muscles were not ablated.

Non-malignant. There were 38 cases of non-malignant tumors, or 37.26 per cent. Women 35, men 3, white 36, colored 2. 27 were married and 8 single. The ages ranged from nine to sixty-six years. The right breast was affected in 16 cases, the left in 21, and in 1 both breasts were involved. The location of the growth is stated in only 16 cases, and of these, 11 were situated in the upper and outer quadrant. The size of the tumors varied from that of a pea to that of an egg or lemon. Three cases had had tumors of the breast removed previously. The lesions were classified as follows: Fibro-adenomata 15; of these 2 occurred in males aged thirty and forty-two years, respectively; fibromyxoma 1, in a male, aged sixty-six; intracanalicular fibroma, 1; cystic fibro-adenomata, 20; chronic hyperplasia, 1.

Two cases were operated on under the supposition that they were adenocarcinomata, but the microscopic examination showed them to be fibro-adenomata. A negro girl, aged nine years, who had a considerable enlargement of the left breast, but not of the right, was supposed to have a fibro-adenoma and the breast was amputated, but the pathological report was "chronic hyperplasia."

In only 2 cases was there a history of trauma. Most of the tumors were freely movable and there were no glandular metastases. Pain was absent in most cases, but in one attention was first called to the breast by pain, when a lump was discovered. The time the tumor had been noticed by the patient before coming to the hospital for treatment varied from one week to

five years, only six being recorded as having been less than one year in existence.

Conclusions. 1. From a consideration of these and other cases, and from the published statistics of other writers, I am still of the opinion that carcinoma is the predominant neoplasm of the mammary gland and that it occurs in from 60 to 65 per cent of all mammary tumors.

2. While it is probable that breast tumors are coming under observation earlier than was formerly the case, nevertheless, in the hospital with which I am connected there has been no marked difference between the time at which this series of cases came under surgical care and that of the series collected in 1911.

3. Sarcoma occurs in about 3 per cent of tumors of the breast. This condition is malignant and should be treated in as radical a manner as a carcinoma.

4. Non-malignant neoplasms occur in about 35 per cent of all cases. In my opinion no woman should be allowed to harbor a neoplasm in her breast, whether it be benign or malignant, or whether she be young or old. Sooner or later benign tumors tend to become malignant, and the time to remove them is while they are still benign. One woman in this series had had a tumor in her breast forty-nine years.

5. In my opinion it is unsafe to trust to the macroscopic diagnosis of tumors, however skilled one may be; and that a microscopic examination of frozen sections should be made at the time of operation of all neoplasms of the breast of which there is any doubt as to their innocency or malignancy.

6. Should the breast of a child approaching puberty be amputated for a uniform enlargement of that organ or should we delay operation until it is evident that the enlargement is or is not due to physiological conditions?

7. The question of the proper treatment of primary or secondary tumors of the sternum is open to debate. Should extirpation be undertaken or should they be treated with radium or *x*-rays?

Quénu and Longuet[7] say in regard to secondary tumors of the sternum: "These interest the surgeon but little; it is evident

that he ought not to touch a lympho-sarcoma of the mediastinum involving the sternum, and the same is true of cancers of the breast involving the sternum. Nevertheless, one may be induced, by necessity or by foresight, to attack the sternum when in the course of an operation for cancer of the breast or of a recurrence of the cancer a sternal adenopathy is discovered."

Finney[8] also says: "Whether it is ever justifiable to open the anterior mediastinum or to remove ribs and parietal pleura cannot be settled arbitrarily."

Rodman,[9] while condemning partial operations for cancer of the breast as worse than useless, advocated very radical operations in some instances. In one case he excised a portion of the sternum and several ribs with a part of the parietal pleura. The woman recovered from the operation and lived nearly three years afterward. Dr. Emmett Rixford,[10] in an excellent paper read before this association in 1905, reports three personal cases in which he excised portions of several ribs and portions of the sternum for recurrent carcinoma; and one case of sarcoma of the clavicle, first rib and sternum, with a large mediastinal tumor, in which he resected the clavicle, a portion of the first rib and a part of the upper end of the sternum. In one of these cases three separate resections of the chest were done. All recovered from the operation and life was materially prolonged in most cases.

Herman Mynter[11] reports an excision of the sternum for melanosarcoma of that bone. A girl, aged twenty years, had a tumor, the size of one-half an orange, extending from the second to below the fifth rib. It was of eleven months' duration and there were metastases in both axillæ and in both supraclavicular fossæ. The tumor was immovable, with an indistinct sense of fluctuation. The skin was normal in color and not adherent. November 14, 1890, under ether anesthesia, a "U"-shaped incision was made, convex downward; the tumor was exposed and scraped out and the bone cut away with bone pliers. The pericardium was exposed for six square inches. The highest temperature was 101° and the wound had healed in ten days.

This was neither a secondary tumor of the sternum nor con-

nected with the mammary glands in any manner, but it was apparently a very malignant growth of the sternum, which was successfully removed; though why it was thought wise to do an operation of such magnitude and gravity on a woman who had extensive glandular metastases in both axillæ and in both supraclavicular fossæ I am at a loss to understand.

8. Equally as serious, and even more frequent, is the extension of cancer from the breast to the ribs. What should be done in such cases? A. P. C. Ashhurst[12] says: "Even fixation to the chest wall does not necessarily contra-indicate excision; the portion of ribs invaded may be removed." The only case in which I have removed ribs for recurrent carcinoma terminated disastrously, but I find quite a number of operations, reported by Fellows of this association, that have been at least measurably successful. Thus Rixford, in the article previously mentioned, credits Stillman with the removal of 5 cm. of the fourth and fifth ribs for recurrent carcinoma, with a rapid and uneventful recovery, the patient being in perfect health two years subsequently. Similar operations have been reported by Dr. Ellsworth Eliot,[13] Willy Meyer and Lucius W. Hotchkiss with encouraging results. I think, therefore, that, while in most cases of direct or secondary involvement of the ribs no operation should be undertaken, in some cases it is justifiable and proper to resect the ribs and the underlying pleura.

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DISCUSSION ON THE PAPERS OF DRS. BRUCE AND WINSLOW

Dr. Albert J. Ochsner, Chicago:

Referring to Dr. Bruce's paper on "The Cancer Problem," I wish to state that about six weeks ago Dr. John W. Nuzum made a very important report before the Chicago Surgical Society which covered his scientific work extending over a period of five years. Two years ago he located what he considered a cancer microbe, so that for the last two years he has worked continuously upon this problem. In this work he has used over 1200 mice.

He had difficulty with cultures because of the peculiarity of this microbe, and has only recently found a medium in which he can culture the microbe regularly. His report, which will contain all the facts, will be found in a future number of the *Journal of Surgery, Gynecology and Obstetrics*.

We have in our every-day practice an example which may find an explanation in Nuzum's experiments. We know that cancer will grow on the gastric side of the pylorus, where there is no bile ordinarily, and it will not grow on the duodenal side of the pylorus, where there is bile present practically always. His pure cultures will grow luxuriantly in ascitic fluid under proper conditions, but they will not grow if this fluid contains bile.

There are a number of other things in which his observations correspond to what we know about cancer at the present time. He takes pure cultures of this microörganism and injects it into mice and a tumor forms, but in most mice that tumor will be absorbed. It seems that the tissues of a healthy mouse have sufficient antibodies to prevent the further growth of this tumor; but if he reënforces his inoculations by further injections of pure cultures so that he overcomes these antibodies then he is able to produce a typical cancer, and this cancer will form metastases and kill the mouse.

Then there are other facts which correspond to what we know clinically of cancer, for instance, if you take one of these cancer nodules, which will grow very slowly, and cut it in two and remove one-half the other half will grow rapidly. If you crush one of these cancer nodules it will grow very rapidly. We know that with a melanotic nevus if we cut into or across that nevus in a short time we will have a diffuse melanotic sarcoma.

Since Dr. Nuzum made this report six weeks ago he has produced cancer in mice into which he has injected pure cultures of what seems

to be the same microbe which he has taken from a cancer of the breast on which I operated, a woman who was about to die from metastases of this cancer. He has produced the same kind of a cancer that he produced from pure cultures of microbes which he took from mouse cancer. He has also taken the pure culture of this same cancer of the breast from this woman and has injected it with her consent at a distance from the location of the former cancer, and there he has again produced a typical cancer.

We have here, undoubtedly, the same microbe which has been reported many times before, but the failure occurred in the inability to culture it; and now that we have a method of culturing it, of course the further studies will go on. To my mind the tumors are typical cancer and the whole series of experiments has covered a period of daily work for two years and the consumption of 1200 mice, so that the work has been done very thoroughly. The results of this work have convinced me that Dr. Nuzum has actually found the cause of cancer.

Dr. Alexander Primrose, Toronto, Canada:

I wish to protest against the value placed upon "quick section" at the time of operation. The fact that it is overestimated was brought home to me at a recent examination of students when most of the candidates, on a written paper, expressed the view that every doubtful case of breast tumor can be cleared up by making a quick section at the time of operation. Such views are undoubtedly erroneous.

The importance of making a careful clinical examination of the patient's condition must be emphasized. One cannot be too painstaking in investigating the local manifestations of the tumor and the constitutional symptoms and history of the patient. After a fairly wide experience I have been impressed by the remarkably small error in diagnosis which exists when clinical methods alone are employed. There are unquestionably a certain number of cases in which we are doubtful in our diagnosis and in which a quick section may be of undoubted value. I use the expression "may be" in the last sentence because even in the hands of the most expert histologic pathologist a quick section may fail to demonstrate cancer when it is present. Let me illustrate by reference to one of my experiences, which, although it is only a single instance, is sufficient to prove my point. I had made a diagnosis of malignancy in one of my patients who had a tumor of the breast. I performed a radical operation without quick section, removing the breast and the axillary contents, muscles, etc., *en masse*.

Subsequent to operation I examined the specimen in the gross and I placed the breast in preservative fluid in one jar and the axillary contents with the glands in another. The jar with the axillary glands was misplaced for a time and the pathologist informed me the breast showed "chronic mastitis" and "no malignancy." I asked him to make many sections, which he did, with the same result. Subsequently the jar with the axillary glands turned up and were found to be the seat of cancer development. He now made a still more thorough examination of the entire breast tissue and eventually found a focus of unmistakable cancer formation. In this case a quick section would almost certainly have failed to establish the diagnosis, and yet the clinical findings were correct in concluding that cancer was present. A further objection to quick section in all cases is the fact that the cancer may be disseminated by the manipulation necessary to obtain material for the quick section. I believe this is far from being a mere theoretical danger.

My practice is to avoid the employment of a quick section in every case in which I can make a reasonable diagnosis of cancer from the clinical examination. I, however, employ a quick section in all cases where I consider I have excluded cancer by the clinical examination but in which I think the possibility of error still exists.

Dr. Frank E. Bunts, Cleveland, Ohio:

I am interested in one point in particular of which Dr. Bruce spoke, and that is in regard to the use of the *x*-ray either before or after operations for cancer of the breast.

Sometime ago I felt that the use of the *x*-ray was absolutely essential in all cases following breast amputations for carcinoma. I am satisfied that in many cases it does a great deal of good and equally so that in others it does an immense amount of harm. The reason, it seems to me, is this, that we have not fully mastered the therapeutic use of the *x*-ray. We know that irritation tends to stimulate the growth of cancer and consequently a dose of the *x*-ray which is not a therapeutic dose will stimulate it and tend to increase the growth rather than delay it. Now I am much more worried about using the *x*-ray after amputations for carcinoma than previously. In the use of the *x*-ray for deep penetration, of course, there is great danger of burning the skin with the *x*-ray, but we all know that experiments are going on in laboratories whereby in all probability we will be able to use a current of perhaps 200,000 volts for deep penetration without burning the skin, and when that time comes we will feel that we have a very valuable adjunct at least in the treatment of heretofore inoperable recurrences of carcinoma.

In the removal of extensive carcinoma of the breast it seems to me it is wise, perhaps, in order to relieve suffering, to have an *x*-ray of the lung taken, for often that will reveal a great many cancerous nodules in the lung which show that operation is entirely devoid of permanent benefit.

I have been greatly interested in cysts of the breast or non-malignant tumors. That matter has been discussed before this Association a number of times, and before other associations a still greater number of times.

I thoroughly agree with the remarks of Dr. Primrose that we cannot depend upon frozen sections. I think that frozen sections can only cover a very small portion of a growth and the pathologist is quite as liable to get that portion which does not contain cancer as that which does contain it, so if there be any doubt I believe our clinical judgment and our macroscopic view of it is more important than the pathologic, and we should not hesitate to do a radical operation if macroscopically or clinically it gives us the impression that it is malignant.

I have been investigating a series of cases which we have had. I have not finished it yet. I have gone over 800 cases of tumors of the breast in which 125 are known to be non-malignant tumors. I have been trying to get answers as to recurrence in the so-called non-malignant tumors upon which we have done partial operations. Thus far I have received only a little over 40 per cent of answers, so that it does not give a large number on which to base an opinion so far as recurrence or lack of proper judgment is concerned.

I have one interesting observation, and that is of a patient in whom I removed a cyst of the right breast, believing it to be non-malignant. I found some two years afterward a carcinoma of the left breast and no recurrence whatever of the right, and it was reported by the pathologist as a cyst, so it is possible that it was cystic at the time and was overlooked and had undergone malignant degeneration, while in the other breast in which the cyst was removed no malignancy occurred.

Dr. Howard Lilienthal, New York:

I am interested in the papers on malignant tumors, and for the most part in accord with what has been said, but I wish to say that last summer, when I heard Dr. Finzi, of St. Bartholomew's Hospital, London, say that following a single therapeutic radiation with the *x*-ray the danger of metastasis during operation was minimized, I have been persuaded to use that method in every case, especially in the breast cases before operation. I have the patients receive one

therapeutic dose and then operate. It seems to me that you are certainly not doing any harm, it will hardly stimulate the cancer, and if it is going to minimize the metastases following an operation I think we should use it.

I also wish to state that I have not lost faith in Coley's fluid in properly selected cases, and that I do use it in the inoperable cases. The inoperable cases have not much to lose, and they can be kept comfortable with morphin. A sufficient number do get well to make it worth while. I hope sincerely the Fellows of this Association will not give up the use of Coley's fluid in the inoperable cases.

Dr. John B. Deaver, Philadelphia:

That all surgeons are not in accord on this subject is self-evident. Personally, I have had some cases treated with the *x*-ray before operation. It may be that the treatments have been too intense, but the wounds have not done as well as in the cases without the preliminary *x*-ray. I have also had one or two instances of ulceration following burns from the *x*-ray.

I can go back many years and recall many instances in which the patients remained well for an indefinite length of time in which we did not use the *x*-ray. I do not believe that the *x*-ray and radium should be used primarily in the treatment of carcinoma. But it is my practice to have the *x*-ray used in all cases after operation, and I believe with good results. I think much harm has been done by the specialists, particularly the gynecologists, and I think their conclusions have been wrongly drawn. I do believe after operation and in the inoperable cases radium should be used, because it is palliative, it prolongs life, and it not only makes the patient more comfortable but also the family. It is a question, however, where the *x*-ray can be used to the same advantage, whether it be better than radium, because the radium confines its action to a smaller space; it is like a torch-light while the *x*-ray is a search-light.

I cannot agree with Dr. Primrose relative to the frozen sections. I believe in making a frozen section, particularly where the operator is a young surgeon, rather than do an unnecessary radical operation without it. A trained surgeon who has seen many cases can in 19 out of 20, if not 49 times out of 50, when sectioning the tumor make a correct diagnosis of carcinoma. At the Lankenau Hospital, Philadelphia, our frozen sections have proven correct in 94 per cent of the cases. I believe every hospital should have a frozen-section apparatus from which a report can be obtained in a short time. These are my views, and I believe they are sound.

Dr. Alexis V. Moschcowitz, New York:

I am somewhat surprised to hear so many of my colleagues discussing numbers of recurrences after operations for carcinoma of the breast, because my experience is totally different. Ever since I learned how to operate properly for carcinoma of the breast (and that is a matter of about twenty-five years), and more particularly in those cases in which at the termination of the operation I was satisfied that I had done the operation properly, I have not encountered a single "local" recurrence.

By this statement I do not wish to imply that all my cases are well. A great many patients have died, but they died of metastases and without any local recurrence. This being the case I believe I can safely claim that they have already had the undiagnosticable beginnings of their metastases at the time of the operation.

Dr. Willy Meyer, New York:

It seems to me that the procedure of exploration of the breast in doubtful cases for the purpose of first making frozen sections is to be condemned. From what I have seen personally I hold to the belief that it is not to the best interests of the patient to enter the so-called "enemy's camp" in order to take out a small portion of the tumor and submit it to a hurried microscopic examination by the pathologist. I have two cases in mind where this was done at the urgent request of the attending family physician and the patient's family against my advice and better judgment. I have always regretted that I submitted to their desire. Further development proved that we had to deal with a cancer of most malignant type; ultimately both patients died. Besides the required rapid microscopic examination sometimes is unreliable. In one of my cases with ordinary ablation of the breast the very last of many cross-sections showed beginning carcinoma; of course, the radical incision was promptly added. I therefore consider the exploratory incision of the breast tumor a bad practice. In cases of doubt we should rather do a radical operation at once, and the young surgeon should perform that as well as the experienced surgeon for the best interests of the patient.

Dr. Randolph Winslow, Baltimore, Md. (in closing):

I will simply say that I am of the opinion that for most of us the best thing is to have a frozen section made at the time of operation.

SO-CALLED CONGENITAL DISLOCATION OF THE SHOULDER (POSTERIOR SUBLUXATION)*

By ALFRED S. TAYLOR, M.D.
NEW YORK

It is my belief that there is no such condition as congenital posterior dislocation of the shoulder.

Inasmuch as a number of isolated cases have been reported in various journals under this heading, and especially since one well-known surgeon and author has, within the last few years, published a series of papers attempting to prove that birth-palsies of the upper extremity are due to such a congenital posterior displacement of the shoulder, it seems worth while to present the evidence against the occurrence of such a lesion and to try to account for the deformity existing in the cases so reported. It is a striking fact that in each of the cases reported as congenital dislocation there has been evidence of disturbance of the motor-nerve supply of the extremity. This disturbance has varied in severity and permanence in the different cases exactly as in birth-palsies of the Erb type.

Therefore it is the object of this discussion to determine whether the disability of the extremity is due to primary injury of the shoulder with secondary involvement of the nerves, as is maintained by Thomas, of Philadelphia,[5] and his followers,[6] or is due to primary injury of the nerves entering into the brachial plexus.

Against the occurrence of posterior dislocation of the shoulder at the time of birth are the following facts:

There is no recorded case in which a posterior dislocation of

* Read by Title.

the shoulder was found at birth. Since 1914, when Thomas read his paper at the New York Academy of Medicine, I have been looking with special care at shoulders in these cases and have never found any degree of posterior displacement in a child less than six weeks old. Unfortunately these cases are not usually referred for a surgical opinion until they are several weeks or months old. During the period January, 1914, to April, 1921, there were in my private files records of 70 cases of birth-palsy, in all but 2 (Cases 9 and 45) of which the condition of the shoulder was carefully noted. Of these 68 cases 22 showed not the slightest degree of posterior displacement of the shoulder. Their ages varied from two days to nineteen years. In the remaining 46 cases posterior displacement was present in varying degree. Not one of these 46 cases was less than six weeks old and 45 of them were eight weeks or more old. Of the whole 68 only 12 cases were six weeks or less in age; 4 were six weeks old, of whom 1 showed posterior dislocation. In not a single one of the remaining 11 was there any degree of posterior displacement. Diligent inquiry among the obstetricians in three of the large lying-in hospitals in New York City elicited the fact that not one of them had ever seen a posterior subluxation of the shoulder *at the time of birth* in any one of the birth-palsies of Erb's type which had occurred in their services.

Dr. Edward D. Truesdell, connected with the New York Lying-In Hospital, saw every case of birth-injury of whatever type during a period of ten years, involving about 30,000 deliveries, and in all injuries of extremities made roentgenograms at intervals. In a personal communication he states that among the thousands of infants delivered there were many cases of birth-palsy of Erb's type, but that he never saw a single one in which there was any degree of dislocation of the shoulder. During this period he was getting the material for his admirable book on *Birth Fractures and Epiphyseal Dislocations*, so that he examined these shoulder-joints with minute care. He is a well-trained surgeon and therefore a competent observer. Inci-

dentally he states that there was never epiphyseal separation of the upper humerus associated with Erb's palsy in his experience.

Dr. H. McM. Painter, for years connected with the obstetric division of the Nursery and Child's Hospital and the Sloane Maternity Hospital, verbally states that he has never seen a birth-palsy with an associated posterior subluxation of the shoulder *at birth*, and that he has called in another surgeon in every injury case in order to check up his own findings.

A diligent search over a period of seven years has failed to find a single authentic case in which a birth-palsy has been associated *at birth* with any degree of posterior displacement of the upper end of the humerus. Even Thomas, who has built up his theory that "birth-palsy is the result of posterior dislocation of the shoulder, with secondary nerve injury, rather than the result of primary plexus injury," states that he has never happened to see a posterior dislocation present in an infant less than three weeks old. The paralysis, on the contrary, is present in all cases at birth.

From the evidence presented it is fair to conclude that there is no such condition as "congenital posterior dislocation of the shoulder," and therefore it cannot be the cause of the "birth-palsy of Erb's type." The term congenital, in this connection, should be discarded.

Nevertheless, in the 68 children above tabulated (see table) some degree of posterior dislocation of the shoulder was present in 46 (or 68 per cent). The youngest child to show it was six weeks old. There were 60 children in the series six weeks or more of age, and of these 46 (or 77 per cent) showed the dislocation. If no dislocations appear until the age of six weeks, and above that age appear in 77 per cent, the deformity must develop as a sequel to the primary injury causing the paralysis, which is unquestionably damage of the brachial plexus.

The conviction that injury of the brachial plexus is the primary cause of the paralysis results from consideration of the following facts:

The paralysis is present at birth; dislocation is absent.

Table I.

	Date.	Name.	Presentation.	Age.	Side and degree.	Dislocation.	Operation.	Findings.	Remarks.
1	Jan. 30, 1914	Green	V	3 wks.	R++++	0	0		
	June 28, 1917	Green	V	3.5 yrs.	R++++	+	July 2, 1917	v and vi torn; vii cicatricial.	
2	Mar. 14, 1914	Plumb	V	3 yrs.	L++++	+	Mar. 23, 1914	v, vi, vii. Posterior disle. disappeared nine months after operation.	
3	July 7, 1914	Lefferts	V	3 wks.	L++++	0	Nov. 25, 1914	v torn off; vi and vii cicatricial.	
4	Dec. 5, 1914	Kachinsky	?	3 mos.	L+	+	0		
5	Jan. 4, 1915	Earnest	V	8 mos.	R+	+	0		
6	Jan. 18, 1915	Hollander	V	5.5 yrs.	L++	+	Jan. 18, 1915	v ruptured; vi cicatricial.	
7	Nov. 10, 1915	Klein	Br.	6 mos.	R++++ eye	+	Nov. 10, 1915	v almost torn off; vi torn completely; vii damaged; viii and i avulsed.	
8	Jan. 31, 1916	Warner	V	9 wks.	R+++ eye	0	Mar. 1, 1917	Stopped because of hemorrhage.	
						+ one year later	April 4, 1917	v, vi, torn across; no Sever; vii cicatrized; viii and i avulsed; post. disle. gone on May 15, 1918 except on strong effort to elevate arm forward.	
9	May 17, 1916	Joyce	V	10 mos.	L+++++	?	May 17, 1916	v, vi, vii torn and cicatrized; viii and D i avulsed.	
10	Nov. 10, 1916	Whiting	V	3 mos.	R++++ eye	+	0		
11	Feb. 2, 1917	Kilpatrick	V	2 mos.	L+++	0	0		
12	Feb. 12, 1917	Dennis	Br.	2 days	R+	0	0		
13	Mar. 6, 1917	Rigby	?	12 yrs.	R+++++	+	Mar. 12, 1917	v, vi, torn across; Separate 1 cm.; bulbous; vii, two-thirds damaged; viii and i scar pressure.	
14	Mar. 19, 1917	Delprato	V	6 wks.	R++++	0	0		

15	April 27, 1917	Lillicrapp	V	8 mos.	L+++	o	o		
16	June 2, 1917	Barry	V	6 mos.	L+++++ eye involved	+	June 5, 1917	v, vi, vii, viii, D i long scar	Second operation Nov. 18, 1919.
17	Sept. 4, 1917	Christ	V	2 mos.	R+ v and vi	+	o		Made a complete spontaneous recovery with reduction of the subluxation.
18	Oct. 17, 1917	Weaver	V	5.5 yrs.	R++++	+	Oct. 26, 1917	Sever; v, vi about one-half torn; vii one-half torn off.	
19	Oct. 26, 1917	Greenberg	V	4 mos.	R++++ eye	+	o		
20	Dec. 26, 1917	Merila	Br.	6 wks.	R++++	+	April 16, 1918	v and vi torn.	
21	Feb. 1, 1918	Dilber	V	3 mos.	L+++	+	Feb. 8, 1918	v torn off; vi and vii cicatricial viii and D i constricted.	
22	Feb. 12, 1918	Van Dine	V	3.5 mos.	R++++	+	Apr. 11, 1918	v, vi, vii resected; no Sever; two yrs. later almost complete reduction shoulder, spontaneous.	
23	Apr. 6, 1918	Carey	V	$3\frac{1}{4}$ mos.	L++	+	Apr. 6, 1918	cv torn off	Almost perfect recovery.
24	Apr. 30, 1918	Sloben	V	10 days	L++++	o	o		
25	May 17, 1918	Eskwith	V	$2\frac{1}{2}$ yrs.	R+	+	o		
26	June 4, 1918	Porta	V	3 mos.	L+++	+	o		
27	June 17, 1918	Corbally	?	14 yrs.	L++	+	Nov. 30, 1918	C v and vi.	
28	June 17, 1918	Tetamore	Br.	2 wks.	R+++	o	o		
29	June 20, 1918	Fitzgerald	Br.	7 wks.	L+; R++	o	o		
30	June 27, 0000	Silverman	V	5 yrs.	R+++ eye (very little power)	o	o		
31	Aug. 2, 1918	Silvagio	V	2 and 4 mos.	R++++++ eye	+	Dec. 14, 1918	v, vi, vii, viii and i damaged; died of hemorrhage on table.	
32	Nov. 14, 1918	Rosenbloom	V	26 mos.	R+++	+	o		
33	Nov. 15, 1918	Margules	V	$2\frac{1}{3}$ yrs.	R+++	+	June, 1918	Operation done by another surgeon who exposed plexus but did not attempt any repair.	

TABLE I.—(*Continued*)

	Date.	Name.	Presentation.	Age.	Size and degree.	Dislocation.	Operation.	Findings.	Remarks.
34	Feb. 6, 1919	Carlson	V	2¼ yrs.	R +	+	Feb. 19, 1919	C v and vi	
35	Feb. 20, 1919	Zucker	?	19 yrs.	L + + + +	0	0		
36	Mar. 7, 1919	Kalmanowitz	V	2 mos.	R + + + +	+	0		
37	Apr. 23, 1919	Levas	Br.	4 mos.	R + + +	+	0		
38	Apr. 29, 1919	Rubino	V	22 mos.	R + + + +	+	June 2, 1919	v, vi torn across; vii torn almost across; viii torn across i normal.	
39	May 20, 1919	Shusteroff	Br.	7 yrs.	R (spontaneous cure);	0	0		
					L: just shoulder remained completely paralyzed	0	0		
40	May 26, 1919	Nobile	Br.	3 mos.	R + + + +	+	June 7, 1919	v, vi and vii torn across, bulbs; viii and i compressed.	
41	June 17, 1919	Allensohn	V	14 mos.	R + + +	+	0		
42	July 21, 1919	Rifkin	V	7.5 yrs.	L + + +	+	0		
43	Oct. 16, 1919	Nagelsmith	?	16 yrs.	R + + + + + eye	+	Oct. 23, 1919	v torn across; vi "almost;" vii ganglion of root extraspinal; viii and i avulsed.	
44	Oct. 30, 1919	Waldman	V	19 mos.	L + + + + eye	+	Jan. 26, 1920	v, vi, torn apart; vii mostly torn apart; viii and i cicatricial into foramina; no Sever; closed reduction; nine months almost no dislocation remaining.	
45	Oct. 30, 1919	Lotz	Br.	3.5 yrs.	L +	?	0		
46	Oct. 31, 1919	Glants	V	2.5 mos.	R + + + +, C viii and D i. eye	+	0		
47	Dec. 26, 1919	Lonetti	Br.	6 wks.	L + + + +	0	0		
48	Dec. 31, 1919	Bettcher	Br.	7 wks.	R & L + + + (R normal in ten days)	0	0		

49	Jan. 19, 1919	Sately	Br.	9 wks.	L++	0	0			At four months had postr-sublux of shoulder which had disappeared at nine months, with improvement of muscles.
50	Feb. 20, 1919	Johnson	Br.	8 wks.	R+++	+	0			
51	Mar. 12, 1920	Teall	V	9 mos.	L++++	+	Mar. 22, 1920	v, vi, vii 2½ cm. dense scar tissue; viii and i constricted; no Sever; postoperative subcoracoid dislocation.		
52	Mar. 17, 1920	Sable	V	5.5 wks.	R+	0	0			
53	Mar. 24, 1920	Johnstone	V	6 yrs.	R+++	+	Apr. 3, 1920	C v and vi half torn through.		
54	Apr. 8, 1920	Bernd	V	10 wks.	L+++	+	0			
55	Apr. 23, 1920	Post	V	2 days	L+	0	0			
56	May 17, 1920	Cucumber	V	7 mos.	L++++	+	0			
57	June 9, 1920	Everson	V	15 mos.	L+++, C viii and D i, eye	+	0			
58	June 23, 1920	Ackerholt	V	5 mos.	R++++ eye small	Very sl. +	0			
59	Oct. 18, 1920	Conroy	V	5 yrs.	R+, mild	+	0			
60	Oct. 20, 1920	Mauersberger	V	10 mos.	R++	+	0			
61	Oct. 21, 1920	McLoughlin	V	16 mos.	L+++++	+	Mar. 23, 1921	v, vi, vii, viii and D i torn and cicatricial		Eye showed sl. enophthalmos and narrowed palpebral fissure suggesting avulsion of D i and C viii; hand also flabby and contractured.

TABLE I.—(*Continued.*)

	Date.	Name.	Presentation.	Age.	Size and degree.	Dislocation.	Operation.	Findings.	Remarks.
62	Nov. 16, 1920	Weinberg	V	5 days	L + + +	o	o		
63	Nov. 29, 1920	Levine	V	2⅓ mos.	R + + + +	+	o		
64	Dec. 9, 1920	Blackman	V	4.5 mos.	R + + + +	+	o		
65	Jan. 19, 1921	Feltman	V	6.5 mos.	L + + + +	+	o		
66	Jan. 25, 1921	Cooper	V	6 wks.	L + + +	o	o		
67	Feb. 9, 1921	Kirschbaum	V	2 mos.	L + + + + eye involved	+	o		
68	Mar. 1, 1921	Mooney	?	2.5 yrs.	R + +	o	o		Pectoralis major atrophied and not contractured.
69	Mar. 18, 1921	Benedito	V	2 mos.	R + + + +	o	o		
70	Mar. 22, 1921	Osterman	V	14 mos.	L + + + + eye slightly involved	+	o		

KEY

Vertex = V.
Breech = Br.
Unknown = ?
Date = First time seen.
Age = Age at first observation.
Side and degree = + to + + + + + indicating in a general way the degree of disability.
Eye or eye involved = Narrowed palpebral fissure (same side as palsy) slight enophthalmos, and smaller eye, usually indicating injury of C viii and D i close to foramina, or avulsion from cord.
Dislocation = + = Present. (Sometimes absent at first examination and present some weeks later.)
= o = Absent.
Operation = o = Not done.
= When done = Date of operation.
Findings:
v, vi, vii and viii = Cervical nerves.
D i = Dorsal nerve.
"Constricted" = Nerves compressed by scar tissue.
"Sever" and "no Sever" indicate whether or not pectoralis major and subscapularis were divided to get reduction of dislocation.
Torn and cicatrical nerves reseeted and sutured, constricted nerves freed.

The distribution of the paralysis is always characteristic of a *root* lesion rather than a lesion of peripheral trunks after they leave the plexus, which latter would be the case if the paralysis resulted from injury secondary to shoulder dislocation. The distribution of the paralysis in Erb's palsy is so characteristic that a little familiarity enables one to predict fairly accurately which roots are damaged in a given case.

In a considerable number of operations (76 in my private files, including also cases 1903–1914) the lesion in the great majority of cases involved the upper roots of the plexus chiefly, and there was a relatively wide area of normal tissues between it and the region of the shoulder-joint and there was no cicatricial tissue in the axilla. In only a very few cases, in which the entire plexus was damaged, did the resulting cicatricial tissues extend downward to the shoulder-joint region.

Thomas states that no case is on record where, at operation, a lesion of the nerves was found other than cicatricial induration, which he believed was secondary to shoulder-joint injury. In the October, 1905, *American Journal of Medical Sciences* (Clark, Taylor and Prout—Case 3), it was recorded that the outer trunk coming from the junction of the fifth and sixth roots was torn off and displaced downward and inward 2.5 cm. and was adherent to neighboring muscle.

In the series of 70 cases seen since 1914, 25 were operated upon, and reference to the findings shows that in many instances nerves were more or less completely torn across (see table). This type of injury could not possibly result from cicatricial infiltration of nerves secondary to injury of the shoulder-joint (Figs. 1 and 2).

In two cases (Case 43 in the above group and an infant, named Passamero, operated upon at the Babies Hospital in October, 1912) a posterior root ganglion was present in the cicatricial tissue well external to the foramina. This indicated avulsion of roots from the cord.

Through the courtesy of Drs. L. A. Wing and J. R. Losee, of the Lying-in Hospital, the following case is put on record:

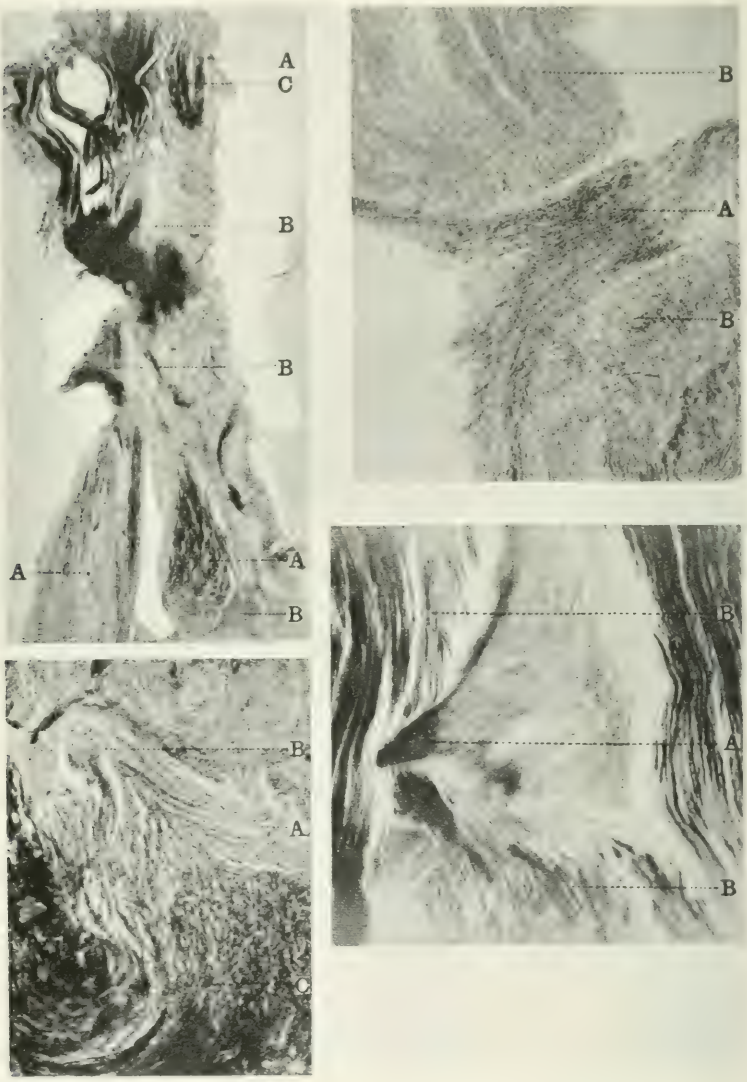

FIG. 1.—These are photomicrographs of nerves resected in the early cases of the operative series. The lesions were all located between the intervertebral foramina and the distal end of the plexus. No mere infiltration of the nerves by material from an injured shoulder-joint (as suggested by Thomas) could possibly cause the results pictured. The very black lines represent nerve fibers more or less damaged and the rest is cicatricial tissue.

Mila, Confinement No. 77,647. Born December 15, 1920. It was a footling presentation with threatened prolapse of the cord. Breech extraction was rendered difficult by a moderately contracted pelvis. No instruments were used. The general

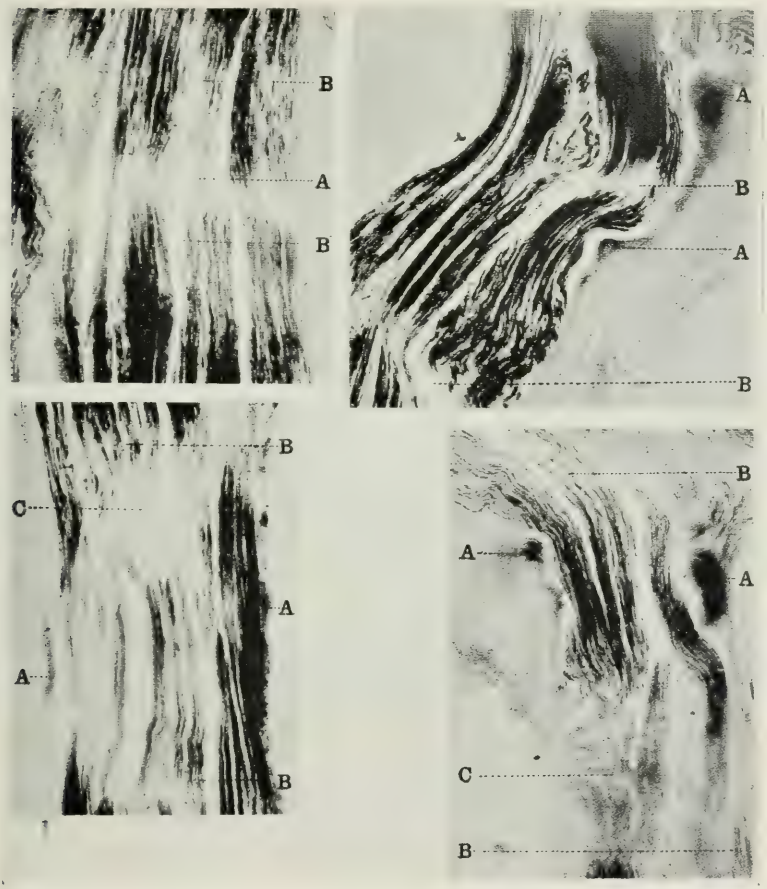

FIG. 2.—These are photomicrographs of nerves resected in the early cases of the operative series. The lesions were all located between the intervertebral foramina and the distal end of the plexus. No mere infiltration of the nerves by material from an injured shoulder-joint (as suggested by Thomas) could possibly cause the results pictured. The very black lines represent nerve fibers more or less damaged and the rest is cicatricial tissue.

condition was good for twenty-four hours, after which signs of intracranial injury appeared and death occurred on the fifth day. At birth, paralysis of both upper extremities was noted. Right side: There was a fracture of the clavicle; the entire extremity showed flaccid paralysis except for very slight movement in the fingers. Left side: There was complete flaccid paralysis.

Autopsy. Showed extensive intracranial hemorrhage with extension of the clot down the spinal canal; fracture of the right clavicle; *both shoulder-joints perfectly normal;* both brachial plexuses macroscopically normal outside the intervertebral foramina; avulsion of the roots of C *iv* and *v* from the cord on the left side. These roots lay outside of the dura (Figs. 3 and 4).

The case of Boyer[1] is equally convincing and more interesting:

On July 4, 1905, during an attempt to deliver the aftercoming head through a contracted pelvis, the right plexus was felt to give way under the finger passing over the right shoulder. A right-sided birth-palsy was prophesied before the delivery, and it appeared in typical form when delivery was completed. No posterior dislocation of the shoulder was present and no injury had occurred to the shoulder-joint. Two hours after birth a a severe convulsion was followed by death. The body was injected with 10 per cent formalin solution and then was immersed in a jar of similar solution. After complete hardening the right brachial plexus was exposed and excised completely from foramina to upper axilla. A typical lesion from overstretching was present at the junction of the fifth and sixth roots. The pathology present was described and pictured in a photomicrograph by Dr. T. P. Prout, in the *Journal of the American Medical Association*, January 12, 1907, xlviii, 96–104.

In 20 plexuses (*Clark, Taylor and Prout: American Journal of the Medical Sciences*, October, 1905), in infants stillborn or dying shortly after birth, typical lesions were produced by forcible separation of the head and neck from the corresponding shoulder. It so happened that in 7 of the 20 the root of C *vi* was avulsed from the cord and the ganglion of the posterior root lay outside of the spinal foramen.

Avulsion in the experimental cadaveric cases occurred in a much higher percentage than seems to be the case in the clinical series. The percentage is difficult to determine in the clinical series inasmuch as laminectomy would be necessary, and is not justifiable, to decide the question (Figs. 3 and 4).

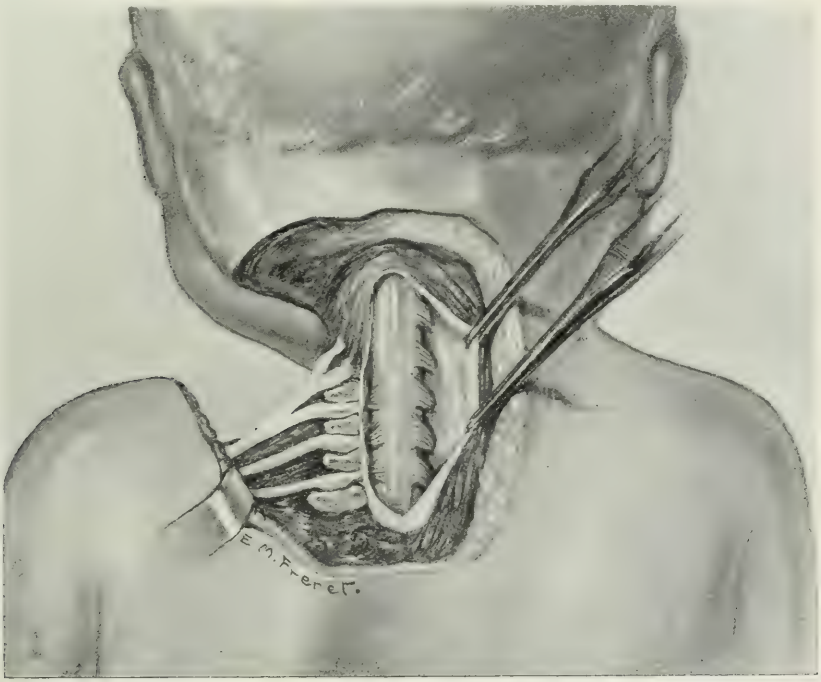

FIG. 3.—The subject was injected with and submerged in 10 per cent formalin solution. The plexus and spinal cord are exposed by dissecting away the soft tissues, removing the posterior vertebral arches and opening the dura. Note all the roots present on the right side of the cord and that two, C IV and V, have been avulsed from the cord on the left side, including both sensory and motor roots. The torn ends of the two roots were extradural. The cord itself was so smooth that the actual site from which the posterior roots were avulsed could not be seen. There was no apparent injury to or deformity of the cord.

These facts taken together prove conclusively that the primary cause of brachial birth-palsy is damage to the brachial

plexus. The etiology, mechanism and pathology were fully worked out in the original publication in 1905, previously referred to, and nothing in later experience has caused a change in the views there expressed.

FIG. 4.—Same infant as in Fig. 3, showing the left brachial plexus apparently intact in its extraspinal portion. The right plexus also appeared normal extraspinally.

Assuming that the primary lesion is a brachial-plexus injury, the later appearance of posterior subluxation of the shoulder in 68 per cent of these children means that there is a distinct cause-and-effect relation between the nerve lesion and the dislocation which must be worked out for the sake of preventive and curative treatment (also see Fairbank[2]).

In the shoulder-joint of the newborn infant the glenoid fossa is very shallow, and in some instances is almost convex; the joint capsule is quite loose (Figs. 6, 7, 8 and 10). Therefore, outside of atmospheric pressure, which plays a minor role, the only structures that hold the upper end of the humerus and the glenoid fossa in their proper anatomic relations are the muscles that pass over and surround the joint. This is the view generally held by anatomists. Through the courtesy of Dr. Lucius

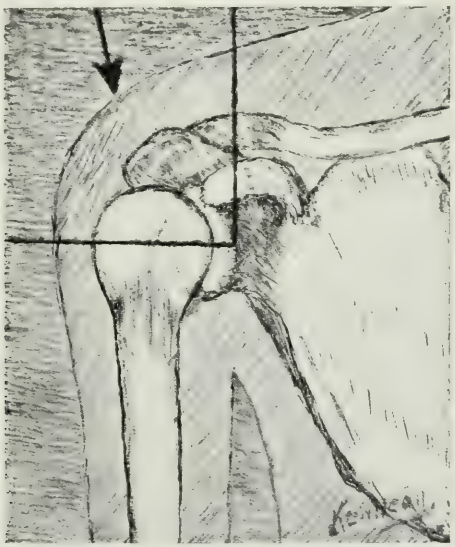

FIG. 5.—Shows the planes of section of the right shoulder in very young infant. The right-angled upper outer portion of the shoulder was removed *en bloc*, giving the exposure for Figs. 6, 7 and 8.

Wing, of the visiting staff of the New York Lying-in Hospital, an interesting demonstration of the above fact was possible. Some twenty infants were examined. When they were asleep, and the shoulder muscles completely relaxed, the upper end of the humerus could be displaced with ease backward, forward, outward or downward for a distance of 1 to 2 cm., but as soon as the infant woke up and resisted, none of these displacements was possible except by the use of undue violence.

If the muscles surrounding the shoulder-joint are responsible for the maintenance of correct anatomical relations between the

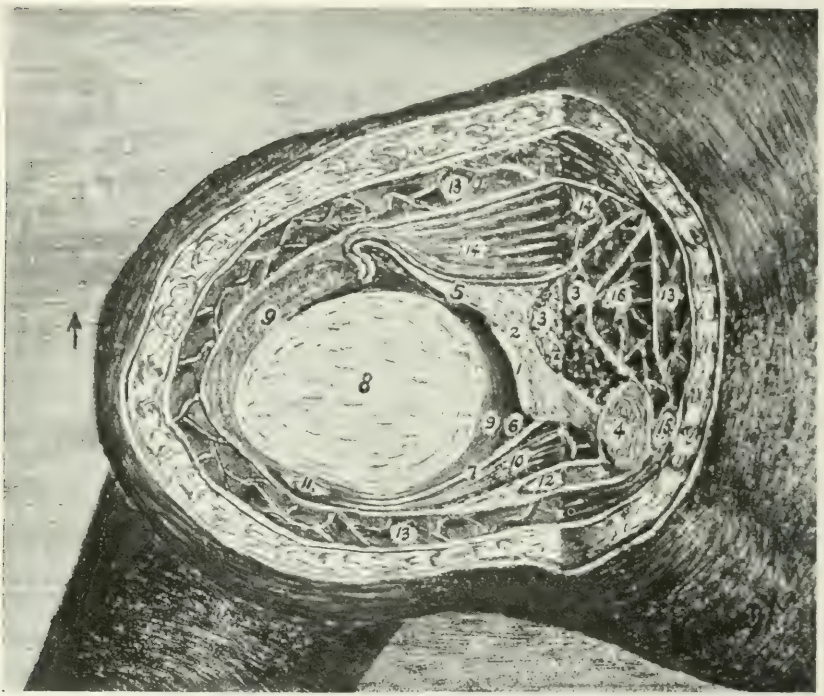

FIG. 6.—The arrow indicates that the humerus is rotated outward, showing relaxation of posterior capsule and muscles. 1, glenoid cavity; note the nearly flat contour. 2, cartilaginous bed of glenoid. 3, scapula, showing ossification; note shapes of cartilaginous bed and ossified portion of scapula. 4, coracoid process; sectioned obliquely. 5, posterior border of glenoid cartilage merging into posterior capsule. 6, inferior portion of capsule. 7 anterior capsule. 8, head of humerus, sectioned horizontally (see Fig. 5). 9, serous surfaces of head of humerus with deltoid bursa externally. 10, subscapular muscle; note that it pulls somewhat backward as well as inward. 11, biceps tendon, long head. 12, biceps, short head, and coracobrachialis. 13, deltoid muscle. 14, infraspinatus muscle. 15, clavicle. 16, supraspinatus muscle.

two bones entering into it, then it follows that the activities of the various muscles must be very nicely balanced against each

other in order always to hold the two bones properly together in the various attitudes of the joint. It would therefore be expected that if one or more of these muscles were paralyzed, while others remained active, the balance would be so disturbed

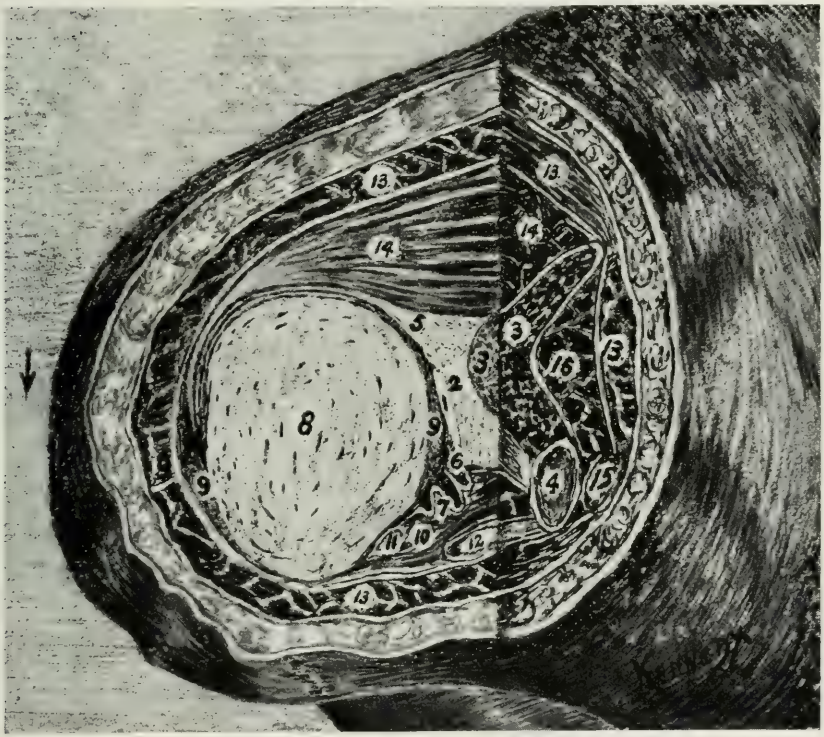

FIG. 7.—Arrow indicates rotation inward of the humerus, showing stretching of the posterior structures and relaxation of the anterior capsule and subcapularis muscle. The portion of glenoid cartilage at 5 is very flexible and causes practically no obstruction to posterior displacement of the head of the humerus.

that deformity of some sort would result. If the muscles involved were only partially paralyzed the disturbance of balance might be so slight that no displacement would result. If all the muscles were partially paralyzed to the same degree there

would be a loss of strength in the joint but no disturbance of balance and no displacement would occur. If all the muscles were completely paralyzed there would be complete loss of power, a flail-joint would result and gravity would determine the displacement.

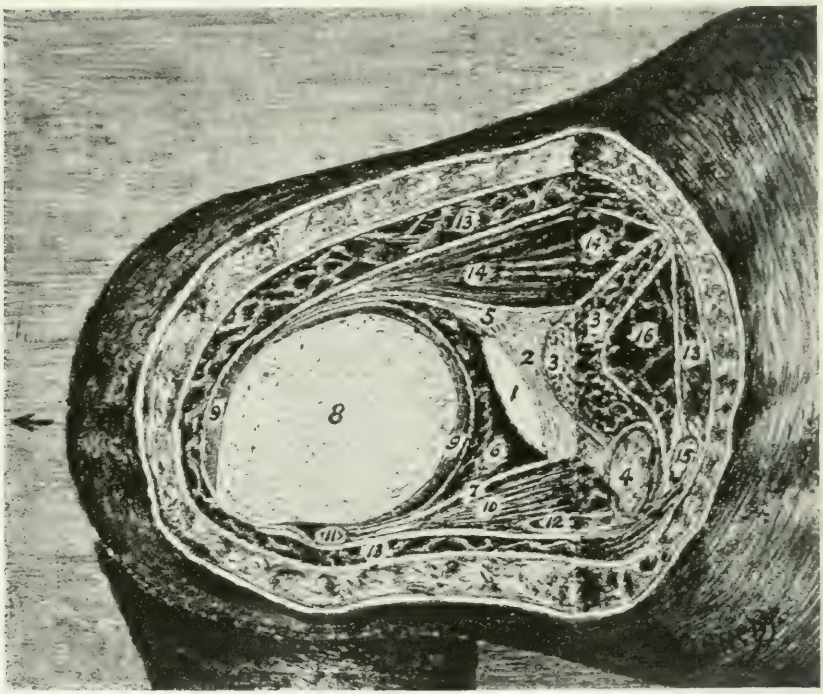

FIG. 8.—Arrow indicates pull outward on head of humerus by manipulating the shaft of the bone. Note the wide separation of the joint surfaces even in the face of rigor mortis. Numbers are the same as in Fig. 7.

In a series of children with birth-palsies one can always find examples of those various degrees of disturbed balance. Except in flail-joint the displacement in nearly every case is backward, quite the opposite of the usual traumatic dislocation of the shoulder.

The most marked dislocations in this series have been observed

in the older children, where complete paralysis of the spinati and teres minor had persisted and where biceps and coraco-

FIG. 9.—The arrows show the effect of pull on the posterior portion of capsule and glenoid cartilage, indicating how slight is the resistance to posterior displacement of the head of the humerus.

brachialis had regained considerable power, as had also pectoralis major and subscapularis, but in the last two there had developed marked contracture.

FIG. 10.—Left shoulder in very young infant with muscles lifted away and head of humerus pulled outward, showing free displacement without injury to capsule. (The muscles are the chief factors in maintaining the joint surfaces in proper contact in the different attitudes of the extremity.) 1, head of humerus drawn laterally and downward. 2, coracoid process. 3, lateral end of clavicle. 4, acromium. 5, glenoid cavity; anterior margin. 6, tendon of long head of biceps. 7, tendon of short head of biceps and coracobrachialis. 8, tendon of pectoralis minor. 9, tendon of triceps muscle. 10, deltoid muscle turned back. 11, deltoid bursa. 12, anterior portion of capsule. 13, coraco-acromial ligament. 14 superior portion of capsule.

The mechanism of the posterior displacement is as follows: The infraspinatus and teres minor form the chief posterior buttress of the shoulder-joint and, being innervated by the two upper roots of the plexus, they usually suffer the most complete and lasting paralysis. Pectoralis major, teres major and latissimus dorsi, being only partially paralyzed rotate the humerus inward and thus overstretch the paralyzed posterior muscles. Teres major and latissimus dorsi also exert traction backward on the humerus. When subscapularis partly escapes paralysis or regains its function it also causes inward rotation and slightly backward traction on the humerus (Fig. 6). Pectoralis major and subscapularis, being only partially paralyzed and having only paralyzed antagonists, first undergo functional shortening and then organic contracture. The head of the humerus is thus gradually forced backward, overstretching to a still greater extent the paralyzed posterior muscles.

Finally, when the posterior subluxation is present, it is greatly exaggerated during attempts of the patient to elevate the extremity. Pectoralis major, coracobrachialis and biceps, when it has recovered somewhat, carry the humerus forward and upward, but at the same time cause a thrust upward and backward along its longitudinal axis, which visibly increases the subluxation of the head of the humerus.

In a small number of cases it has been stated that, while a well-marked posterior subluxation of the shoulder is present, all the muscles of the extremity react to the faradic current, that therefore there is no persisting paralysis and therefore the dislocation cannot be the result of a paralysis due to nerve injury. Since this apparently paradoxical condition is seen only in children one or more years old it can readily be accounted for on the basis of the mechanism previously presented. The dislocation appears usually in from six weeks to three months and is always associated with marked inward rotation of the humerus and with contracture of the pectoralis major and subscapularis. These contractures are usually so marked that the humerus cannot be rotated outward passively, even by the

use of much force, so that the flexed forearm will come to the sagittal plane, *i. e.*, less than 50 per cent of its normal. Therefore the external rotators are continuously overstretched, and even if their nerve supply regenerates sufficiently to give faradic reaction they nevertheless suffer a constant relative handicap, the muscular unbalance persists and the dislocation remains. If, in addition, the coracoid has grown forward and downward it adds a permanent obstacle to spontaneous reduction.

The persistence or disappearance of the dislocation depends upon whether or not the external rotators can overcome their handicap and whether or not the coracoid interferes. The majority of them persist.

A number of very young infants have been seen with birth-palsy in whom no sign of dislocation was present. After several weeks or months, during which parents have failed to follow advice, they have again appeared with definite posterior dis-displacements. (Warner (Case 8) and Sately (Case 49) in this series.)

One case in this series (Sately, Case 49), first seen at nine weeks of age, showed no dislocation; at four months showed definite posterior displacement; then, going on to a spontaneous nerve and muscle regeneration, returned at nine months with no sign of posterior displacement. This is perhaps the most convincing argument of all that the displacement is the result of disturbing the balance in the action of the shoulder-joint muscles.

In several cases of this series where well-marked dislocation was present before operation, and where at operation nothing except a nerve repair was done, postoperative recovery of the nerves and associated muscles has been accompanied by a spontaneous gradual reduction of the displacement (Van Dine (Case 22), Warner (Case 8), Plumb (Case 2) and Waldman (Case 44).)

It therefore seems fair to deduce from the material presented that there is no such entity as congenital posterior dislocation of the shoulder, and that in the cases so reported the dislocation

is a delayed sequel of a primary nerve injury which has caused an unevenly distributed paralysis in the muscles about the shoulder-joint. The resulting unbalance leads to the development of the dislocation.

Treatment is primarily prophylactic, for this dislocation need not occur if the paralyzed extremity is properly treated

FIG. 11.—Shows an older child held in the desired position by the author's brace. This happens to be a postoperative case and therefore the head is harnessed to prevent traction on the sutures.

from the beginning. Maintenance of correct posture is fundamental, as this prevents both the overstretching of the paralyzed muscles and the contracture of the non-paralyzed or partially paralyzed antagonists. The best posture consists in abduction of the arm to 90 degrees or more; external rotation of the humerus

until the flexed forearm is in the vertical plane; flexion of the elbow to 90 degrees or less; complete supination of the forearm; and extension of the wrist and fingers (Fig. 11). This posture is almost perfectly attained if the palm of the opened hand is held on the vertex of the skull, with the elbow held out in the transverse plane of the body, and may be maintained by any one of a great variety of methods. Moreover, this posture helps mechanically to prevent posterior displacement of the shoulder and causes relaxation of the nerves which have been damaged by overstretching and so favors their spontaneous recovery to the maximum degree. After the first two or three weeks, during which manipulation is very painful, the extremity should be removed from its fixation two or three times a day and given massage and passive motion.

In appropriate cases, at the proper time, surgical repair of the damaged nerves should be done. There is much and heated argument as to what cases are appropriate and what time is proper for surgical repair, and the question is by no means settled. The author's opinion is a matter of repeated record, and is modified by later experience only to the extent that a longer period between birth and operation is permissible provided correct care, as above indicated, is continuously given.

In those cases in which the dislocation has occurred it may be readily reduced by the method of Sever.[3] Nerve repair may be done at the same time. Whitman's method, which consists in stretching rather than cutting the contractured muscles, followed by fixation, is not so satisfactory, as there is a strong tendency to recurrence of the dislocation. Moreover, in many of the cases, the tip of the coracoid process has become elongated forward and especially downward so as to mechanically interfere with complete replacement of the head of the humerus. This elongated tip must be resected subperiosteally to attain the best result. Occasionally it is also necessary to do osteoplasty on the acromion process because of a similar distorted overgrowth.

In any case, whether operated upon or not, the fixation and

physical therapeutics above outlined must be continued until the paralyzed muscles and damaged nerves have recovered sufficiently to prevent the muscular imbalance which would otherwise result in posterior subluxation.

SUMMARY.— Since there is no case on record where, in a birth-palsy case, a posterior subluxation of the shoulder has been found *at birth;* since obstetricians connected with three of the large lying-in hospitals of New York City have never seen a single instance of the association of the two at birth; since, after a search during seven years, the author has never been able to find posterior subluxation in any birth-palsy case less than six weeks old, he is forced to believe that "congenital posterior subluxation of the shoulder" does not occur in birth-palsy cases and therefore cannot be the cause of the palsy.

The term "congenital" should be discarded in this connection.

Since the author's series shows no dislocation present up to six weeks of age, but shows its presence in 77 per cent of the 60 cases six weeks or more old, it is obvious that the dislocation is a sequel of the paralysis.

That the paralysis results from nerve injury is evidenced by these facts:

Nerve lesions have been found and resected in a large number of operative cases (Figs. 1 and 2), where they could not have been the result of injury to the shoulder. The area of cicatricial nerve damage was widely separated from the shoulder-joint structures, which appeared of normal texture except for the contracture in some of the anterior muscles. The nerves were torn across, avulsed from the cord, or shredded and cicatrized.

Lesions precisely similar to those found at operation have been experimentally produced on the infant cadaver.

In one case the plexus was felt to give way under the author's fingers and at birth there was a typical palsy; no disturbance of the shoulder was present, and autopsy showed a nerve lesion precisely similar to the experimental and operative ones.

Figs. 3 and 4 show a distinct nerve lesion in a case of bilateral palsy where neither shoulder showed dislocation or other injury either clinically or at autopsy.

The posterior dislocation is a sequel to the unbalanced paralysis of the shoulder muscles and may be prevented in most cases by proper treatment.

The external rotators (posterior muscles) suffer the most complete paralysis and are continuously overstretched by the internal rotators, which are usually not completely paralyzed and eventually develop organic contracture, with the result that the head of the humerus is gradually displaced backward.

Treatment of the dislocation is:

Preventive. By maintaining correct posture and using physical therapeutics from the time of birth until the muscle balance is sufficiently restored.

Surgical. Nerve repair, when indicated, supplemented by the Sever operation when the dislocation is irreducible. These operations are to be followed by postural treatment and physical therapeutics until the muscle balance is sufficiently restored.

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SOME SECONDARY COMPLICATIONS OF POSTERIOR GASTRO-ENTEROSTOMY

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Since during the last few years the end results of surgery are being studied more and more and the value of an operation is based upon the eventual benefit which the patient has derived, I believe that the following cases of posterior gastro-enterostomy, with their secondary complications and their end results, are of sufficient interest to bear reporting.

Case I.—J. E. T., Raquette Lake, N. Y., aged forty-nine years, United States guide and gardener, entered the Albany Hospital August 5, 1910. Discharged September 2, 1910. Recovered.

Patient states that about seven years ago he had attacks of stomach trouble and was treated by his physician for indigestion. He has had a number of these attacks ever since, averaging about four each year. On July 6, 1910, while at work, he was taken with dull pain in epigastrium extending down to umbilicus; at times the pain would radiate downward toward pubes and also around to back on both sides, but being more persistent on right side. He stopped work on July 8, and went to bed July 13, on the advice of his physician. He has been out of bed since July 18. On July 6 he had an attack of nausea and vomiting. On July 27 he had another attack of nausea and vomiting. He says that his appetite is good but a little food seems to satisfy him. He has never seen any blood in vomitus nor does he recognize any particles of food of previous meals. There is no certain

time after meals for vomiting to recur; says it may vary from four to six hours or more. Patient can eat fruits, tomatoes, but meats seem to distress him. At times he has eructations of acidulous fluid into mouth. Tongue is slightly eroded in middle but red on edges and tip.

Physical examination revealed a small indurated mass in the epigastric region, rather to the right of the median line. No glands palpable in the body. Examination of stomach contents disclosed a small amount of blood, undigested food and lactic acid bacilli. Examination of urine showed a large number of hyaline and granular casts, about 3 per cent of albumin, no sugar, specific gravity 1016.

It being before the time that the technic of the *x*-ray examination of the intestinal tract had been developed, no radiographic examination of stomach was made. From the clinical history and the physical findings a diagnosis of pyloric obstruction was made, probably malignant in character.

Operation, August 10, 1910. A tumor the size of a horse-chestnut, having many characteristics of an inflammatory nature, was found at the pyloric end of the stomach, causing partial obstruction. No lymph glands found involved. A posterior gastro-enterostomy was made. An uneventful convalescence followed, and the patient left the hospital on September 2 apparently well and eating anything he wished. Pathological report of intestinal mucosa was negative. No evidence of malignancy in fragment submitted. Examination of urine upon discharge from the hospital was practically negative. This man reëntered the hospital October 11, 1913, for removal of a prepatellar bursa of right knee. He reported that he had no trouble from his stomach whatsoever and could eat any kind of food without distress.

He again entered the hospital October 21, 1914, complaining of pain and distress in his stomach, and frequent urination. Examination of specimen of urine at that time gave results as follows: No sugar, granular and hyaline casts, acid, 1018, albumin. Smears—no tubercle bacilli or other organisms seen. Cultures—sterile at the end of forty-eight hours' incubation.

The patient admitted having partaken freely of alcoholic stimulants for the past few months.

Under appropriate treatment the stomach and kidney condition cleared up and he left the hospital October 27, 1914, recovered.

He reëntered the hospital October 8, 1918, complaining of distress and pain in the stomach and diarrhea. At this time roentgen diagnosis was: Esophagus normal. In standing position the stomach empties very rapidly through the gastro-enterostomy opening, but some barium is seen to leave by the pylorus. No incisura, filling clefts or notches seen. Cap seems normal. The stomach does not empty so fast through gastro-enterostomy opening in prone position. There is very slight six-hour retention. Barium has reached the descending colon, but most of it is in the cecum; a small amount is in the small intestine.

Owing to the clinical and *x*-ray findings, an exploratory operation was advised. Patient returned home and returned at the end of a week, and entered the Homeopathic Hospital, where the abdomen was reopened through the old scar. Examination of the pylorus, the site of the tumor found at the operation August, 1910, revealed a normal pylorus with absolutely no evidence of any tumor or cicatricial tissue. At the site of the gastro-enterostomy, however, was found a tumor mass about the size of the one previously found at the pylorus. In addition the lymph-glands along the spine were involved. Clinically, the case presented was one of carcinoma. Owing to the poor condition of the patient no tissue for pathologic examination could be removed. The incision closed. The patient is still living, two and a half years afterward. Why did this man not make a complete cure?

There is much doubt as to the case being malignant, but is it not quite probable that it is one of those cases which we are beginning to recognize as a secondary ulcer occurring at the site of the gastro-enterostomy? Where is the fault in this case along the line of metabolism producing a condition tending to

gastric ulcer? This case presents two interesting points. First, the fact that, in cases of inflammatory or malignant disease, where we can stop the irritation of the growth we can either stop the growth and have it disappear entirely, as it did in this case, or else we can so diminish its growth as to greatly relieve the patient, as is shown in cases of carcinoma of the rectum relieved by a colostomy and the patient given comfort for months, if not years.

The second point is the appearance of the growth, possibly carcinoma, at the site of the anastomosis, showing that the irritation of the gastric juice must have set up a new growth entirely independent of the previous growth at the pylorus. Case is now in the hands of Dr. Worrell, who diagnosed it as one of chronic interstitial nephritis.

Case II.—G. Y. H., Jr., aged thirty-six years, single, farmer by occupation, was brought to the hospital on January 11, 1915, in a state of collapse, and with the history that for the past two or three years he had had trouble with his stomach. In the beginning there was only pain in the stomach before meals, which pain was relieved by eating. About three hours after eating the pain would return. He would have a lot of gas on the stomach. Later pain diminished somewhat and the patient would vomit large quantities of food. This vomitus would be of a sour taste, burning when it came up and of a dark brown color. During the night of January 11, 1915, he was seized with severe pain in the epigastric region, and went into a state of collapse. He was operated upon next morning for a perforated gastric ulcer. Closed by use of silk purse-string suture. Drainage with gauze and one rubber tube. Counter-drainage in right iliac fossa. He left the hospital February 4, 1915, apparently well.

He reëntered the hospital March 25, 1915, complaining of pain in the region of old trouble. Since leaving the hospital in February he has some pain, but is bothered with distress in the stomach and would vomit occasionally, the vomitus being light in color. He entered the hospital March 25, 1915, seeking relief from distended stomach. Posterior gastro-enterostomy per-

formed March 26, 1915, from which he made a good convalescence and left the hospital April 14, 1915, feeling absolutely well and able to eat anything in reason.

He remained well for two years, when he commenced to complain of his old symptoms, which would yield at times to appropriate treatment. On February 23, 1919, he was seized with an acute pain in the epigastric region and went into a state of collapse, from which he rallied and the pain gradually localized itself in the region of the appendix. He was brought to the hospital; an incision was made over the region of the appendix, which was removed, slightly congested, but the abdomen was found full of gastric contents. An incision was then made through the old gastro-enterostomy wound and a perforated ulcer found at the site of the anastomosis on the jejunal side. This perforation was closed by a mattress silk suture. Two rubber drainage tubes inserted in upper and lower edges of wound. He made a slow but uninterrupted convalescence and left the hospital March 6, 1919, in good condition. He has remained well ever since, has had no more distress in stomach, is able to eat anything he wishes, and has gained considerably in weight.

The interesting point in this case is the fact of his developing an ulcer at the site of the anastomosis giving very little, if any, symptoms until its final perforation. Also the fact that he made a good recovery from the perforation even though the diagnosis and operation were delayed for twenty-four hours. I am free to confess that I should have made the diagnosis more promptly, but the fact that he had had very slight previous gastric symptoms and that he had had one perforated gastric ulcer did not seem to make it possible that he should have another. As a matter of fact, the possibility of such a condition being present was not even thought of.

That peptic ulcer occurs at the site of a gastro-enterostomy is of course a well-known fact, and numerous cases of that condition are reported in the literature. Various theories are advanced for the same, and the fact that they do occur probably accounts in part for some of the failures to give relief in stomach surgery.

A thorough search of the literature, however, fails to reveal the report of a case of perforation of an ulcer so occurring—though, of course, there is no reason why a perforation could not occur as well in that situation as in a primary gastric or duodenal ulcer. So far as treatment is concerned, I merely closed the perforation by a purse-string silk suture and drained. He has had no subsequent symptoms.

Contrast these two cases with the history of a third patient, Mr. G., aged sixty-six years, upon whom I operated for a stenosis of the pylorus, performing the identical form of operation, who had absolutely no stomach symptoms afterward, lived for fifteen years and then died of pneumonia.

Why should the two cases in the younger patients develop secondary ulcer, and the third case, in a much older man, make practically a perfect recovery?

CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS

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I HAVE chosen the above name to apply to an aggregation of symptoms, the diagnosis of which has often been overlooked and the etiology of which is still uncertain, giving rise to a variety of terms for this condition.

In common with other observers, my experience leads me to believe that in congenital stenosis of the pylorus the hypertrophic and not the spastic element is the more important, and that such terms as "congenital spastic pyloric stenosis," or merely "pyloric stenosis of infancy," are misnomers. This paper, therefore, will deal only with those cases characterized by some degree of hypertrophy of the pyloric muscle bundle with associated obstruction, partial or complete—the type which generally requires surgical intervention for permanent cure.

Although the condition has been receiving widespread attention only within the last few years, isolated cases have been reported since 1788. At that time, probably what is the earliest known observation was reported to a medical society in Connecticut, by Hezekiah Beardsley, who, in describing the pylorus, states: "The pylorus was invested with a hard, compact substance, or scirrhosity, which so completely obstructed the pastage into the duodenum as to admit with the greatest difficulty she finest fluid."

But it is only since the latter part of the nineteenth century,

as more cases began to be reported, that physicians began gradually to recognize the condition as a clinical entity and one of comparatively frequent occurrence. Diagnosis and treatment have accordingly become more accurate, with a corresponding decrease of mortality.

Etiology and Pathology

The disease occurs in the earliest infancy, the symptoms coming on within a few days to several months after birth; generally in the second or third week. The infants, as a rule, are well developed and apparently normal at birth, the majority being first born, of the male sex, and most frequently breast fed, so that indigestion from faulty feeding plays no part in the etiology of the disease. In fact, no satisfactory explanation of the cause has as yet been forthcoming.

While spasm of the pylorus may occur at any age, it does not produce the condition of hypertrophy peculiar to this disease of early life. The recovery under medical treatment of certain cases of pyloric spasm, mistaken for true hypertrophy, has led to overestimation of the value of medical treatment for hypertrophic pyloric stenosis. According to Holt,[1] persistent spasm of the pylorus does not exist without hypertrophy, and the use of the term "pylorospasm," he believes, is responsible for loss of life which might have been saved by operation. He regards hypertrophy, undoubtedly congenital, as the essential lesion in all cases; while a definite element of spasm may be present, he believes it to be the result and not the cause of the hypertrophy. This would explain the case in which Dent found pyloric hypertrophy in a seven months' fetus, as well as the presence of the tumor when found in stillborn infants.

Holt divides the cases into mild and severe ones, while Cautley[2] distinguishes those in which muscular hypertrophy and redundant mucosa are so marked as to cause absolute anatomical stenosis, and those in which hypertrophy, although present, has caused only slight narrowing of the pyloric passage. Obstruction in the

latter type being due to irritative spasm of the hypertrophied pyloric sphincter, recovery under medical treatment is possible.

Downes[3] explains the sequence of events in the disease as follows: "True malformation is present at birth, consisting of an abnormal thickening of the circular muscle of the pylorus, and the effort to force food through the narrowed and elongated pyloric lumen produces circulatory disturbances resulting in edema. As the food increases in amount and the muscular effort becomes greater, the lumen narrows down until finally at the tenth day or a little later it becomes more or less completely obstructed." In support of this theory he calls attention to the fact that: "After symptoms have developed, a reduction in the amount of food with consequent relief of muscular effort, together with systematic stomach washing, which removes curds and mucus, will often give temporary relief and in exceptional cases, if the hypertrophy is not too extensive, carry the child along for a time. However, as the food is again increased, all symptoms recur."

Strauss[4] believes that the condition begins during fetal development of the stomach, and is brought about by rhythmic contractions of the pylorus, which doubtless start at that time and are due to abnormal stimuli from intrinsic or extrinsic nerves of the stomach. These independent rhythmic contractions of the pylorus produce hypertrophy on the same basis that any muscle that is in constant motion becomes enlarged. The condition must then either progress very slowly or scarcely begin during fetal life. It becomes accentuated after birth, owing to the additional irritation produced by the taking of food. The tumor gradually increases in size, as a result of the rhythmic contractions of the pylorus, until at the age of seven to twelve weeks the tumors are four or five times as large as those found at the third or fourth week. The size of the swelling, however, does not indicate the amount of obstruction present. A large tumor may be associated with very little pyloric obstruction and a small one may produce almost complete obstruction.

In passing it may be said that attempts have been made to

associate pyloric stenosis with the occurrence of other conditions, such as enlargement of the thymus and phimosis, but conclusive observations to this effect are still lacking. Gray and Pirie,[5] by a complicated theory, conclude that "congenital pyloric hypertrophy is the result of prolonged antenatal spasm induced by hyperadrenalism," and believe that an adherent prepuce or phimosis may act as a source of irritation to the adrenals, the consequent hyperadrenalism possibly leading to hypertonus of the pyloric sphincter, whether a tumor be present or not.

In true hypertrophic stenosis there is always a tumor present at the pyloric ring. When found at operation or postmortem, it appears as a firm oval or cylindrical-shaped swelling, roughly one-half to three-quarters of an inch long and covered with smooth, glistening, white peritoneum. There is no external evidence of inflammatory reaction and no attempt at fixation. The tumor has the firmness of cartilage, is never soft or friable, and is always sharply defined, particularly at the duodenal end. It is formed chiefly by hypertrophy of the circular muscular fibers, or normal histological structure. The mucous membrane is folded up, more or less thickened, and may be spirally arranged; the submucosa is little, if at all altered. The lumen is small and the stomach is dilated, and in cases of long standing may show a diffuse hyperplasia of its muscle. Although these tumors are but simple muscular hypertrophies and there seems no obstacle to their ultimate disappearance, the mass seems to persist, at least for several years. Surgical treatment by the Rammstedt pyloroplastic operation, however, has proved effective in restoring the normal anatomy of the pyloric region.

Downes, March and Ibrahim,[6] and others, have proved by postmortem findings on children who have died of other ailments, after recovery through medical treatment, that the tumor persisted. Quite a number of cases subjected to gastro-enterostomy have been collected by Holt, showing the persistence of the tumor for years afterwards, as demonstrated by subsequent operation or necropsy. The tumor was merely sidetracked. Eahleson showed that a bismuth meal traverses the anastomotic opening and not the pylorus.

On the other hand, Downes[7] reports autopsy findings in two patients who died of pulmonary conditions a year and a half after a Rammstedt operation. In both instances the pyloric tumor had entirely disappeared, and with the exception of light omental adhesions at the site of the scar the pylorus appeared normal in every way.

Ransohoff and Woolley[8] made sections of the pylorus of an infant who died at six months of age of thymic death, on whom a Rammstedt operation had been done when ten weeks old. They say: "It appears that the surgical incision permitted the muscular coats of the tissue to retract and the opening to gape, and that subsequently the space was filled with organized tissue which in its fully developed state was fibrous tissue. This had formed a typical, linear, contracted scar. It seems that during the healing of the wound the pyloric orifice became dilated and then remained so, and thereafter performed its function in an acceptable manner. This is anatomic proof of the perfect efficiency of the operation. It shows also the futility of the numerous modifications which only complicate the operation without in the least improving it. Another interesting feature is the disappearance of the tumor mass which was plainly shown at operation."

Gray and Pirie[5] quote Rachford's postmortem findings six months after the Rammstedt operation. (Death from another cause.) In this case, contrary to his experience after gastroenterostomies, no trace of the tumor could be found. It has also been the experience of these authors, both clinically and at postmortem, that after a Rammstedt operation the pyloric hypertrophy rapidly disappears.

Clinical Features of the Disease

The diagnosis of hypertrophic stenosis of the pylorus is based chiefly on the clinical picture of—

(*a*) Persistent projectile vomiting in an infant a few days to several weeks old, and more frequently in first born, breast-fed infants of the male sex.

(*b*) A palpable tumor in the region of the pylorus.
(*c*) Visible gastric peristalsis, and
(*d*) Gastric retention.

In addition, one notes the absence of milky stools, emaciation and dehydration, the dilated stomach and the crying from hunger and pain. Although some cases cannot be diagnosed at the first examination, a few days' observation will usually make the recognition easy.

Strauss considers *x*-ray and fluoroscopy most important for accurate diagnosis. He insists upon its use in every case, not only for definitely showing the presence of the congenital hypertrophy but also for indicating whether treatment should be medical or surgical. If at least 70 per cent of the bismuth meal does not pass through the pylorus within four hours, operation is indicated.

According to Arens,[10] the *x*-ray picture comprises: Dilated stomach; hyperperistalsis; characteristic snakelike movements of the antrum and pylorus; partial or total obstruction of the pylorus; one-third or more, four-hour retention of the bismuth meal.

Holt, on the other hand, regards the use of the *x*-ray unnecessary for diagnosis and even objectionable because of the possibility of increasing pyloric obstruction by the bismuth.

Downes, while advocating the use of the *x*-ray, does not resort to it in the average case because of the delay in operation it entails, which even though it be only a few hours may be dangerous in some cases.

The vomiting of pyloric stenosis must be distinguished from that of gastric indigestion, gastritis or simple regurgitation. Gastric indigestion, though a common complaint of infancy, is not frequent in the nursing infant and rarely develops suddenly. Temporary pylorospasm due to faulty feeding may undoubtedly exist in early infancy, but the signs and symptoms are milder, there is less visible peristalsis and the condition is more amenable to treatment. Other rare conditions requiring differentiation are tumors or adhesions around the duodenum which produce

stenosis or kinking, congenital atresia of the pylorus or malformations and atresia of the small intestine.

As a rule, the symptoms of a case of congenital hypertrophic stenosis are very definite and with few exceptions diagnosis can be made from the clinical picture. The chief early sign is sudden abrupt vomiting of food, without any apparent cause, in a previously healthy infant. Vomiting persists in spite of changing the food, and soon becomes copious and explosive, occurring two or three times a day. The child at first does not appear sick, and there is no evidence of gastritis or tenderness over the stomach, and the food on reaching the stomach does not act as though it met an inflamed mucosa. When the stomach is nearly empty the food may be retained for a while, only to be vomited later when additional food is taken. The vomitus is bile-free and copious, according to the degree of obstruction. When gastric retention is marked, the dilated viscus may be seen extending to the navel; but generally the stomach is not distended, owing probably to the constant vomiting. If the obstruction is incomplete, the infant may retain a part or all of occasional feedings and fecal stools may persist. Emaciation is less rapid in these cases. While a minority of them recover after weeks and months of a precarious existence and without operation, or the so-called medical treatment, pyloric stenosis usually results fatally in from six to ten weeks. In the greater number of cases the stenosis will be so pronounced that constipation becomes absolute and the stools are present only as bile and mucus in the form of a dark-green paste resembling meconium. Diarrhea with green, watery stools is rare. The emptiness of the intestines gives a characteristic sunken appearance to the lower half of the abdomen. Abnormal gastric retention is one of the diagnostic features of pyloric stenosis—food being almost invariably found in the stomach after three hours, and oftentimes the amount vomited at one time is considerably greater than the amount of food just taken. Holt recommends determining the amount of gastric retention by means of suction instead of the *x*-ray.

Normally a wave of peristalsis passes over the stomach toward

the pylorus about once every twenty seconds, but these waves are invisible. When pyloric obstruction exists and the baby has just taken some food or water, exaggerated peristaltic waves become distinctly visible, if looked for in a good light, a well-exposed abdomen and a quiet baby. Slight manipulation or tapping of the epigastrium may often excite the waves. They appear as ball-like tumors moving slowly and regularly across the abdomen from just below the ribs on the left, to flatten out and disappear on the right side beyond the median line. Typical gastric contractions can hardly be mistaken for anything else and repeated examinations under favorable conditions should be made for this almost pathognomonic sign of hypertrophic stenosis of the pylorus.

Of great importance is the presence of the pyloric tumor, which can with experience be felt in most cases. Downes considers the presence of the tumor as characteristic of the disease and states that its absence is exceptional. In only 2 of his 217 cases did the anteoperative diagnosis of a tumor at the pylorus prove incorrect. Typically it appears as a hard finger-like lump about three-quarters of an inch long, which is movable downward and sometimes may be felt to contract and to change in hardness during peristalsis. Variations in its size may account for the occasional difficulty in finding it, the smaller ones naturally presenting the greater difficulty. Before palpating for the tumor, excessive gas in the stomach should be relieved with a stomach tube. Palpation of the mass usually is easier during active peristalsis. It is felt about one-and-one-half to two inches below the free border of the ribs just inside the right mammary line, but it may be obscured by distention of the stomach or the colon, or by an enlarged liver overlapping the pylorus. Sometimes it may be difficult to get the examining finger behind the lower border of the liver to palpate deeply enough, while in older children of two or three months the better-developed abdominal muscles increase the difficulty of palpation. Moreover, the coils of intestine are somewhat distended, as they have been functionating longer. Some authorities make the examina-

tion under light anesthesia in order to quiet the child and overcome muscular rigidity, but usually patience and putting the patient in a warm bath is safer and also sufficient to detect the tumor mass.

The emaciation is a striking feature of this disease. In the severe forms which are little influenced by medical treatment the loss of weight is progressive and may amount to three or four ounces daily. A fatal termination from starvation may ensue in a few weeks. In the milder form, with less-marked symptoms, vomiting is less frequent and the weight remains more or less stationary; but it is not until after several months, if the infant is not cut off by intercurrent disease, that convalescence begins, as noted by increased weight and the disappearance of digestive symptoms. Even then the peristaltic waves and the palpable tumor may persist until the end of the first year, while evidence of incomplete obstruction may continue for months or years.

A particular feature of hypertrophic stenosis which has attracted my attention is the extreme dehydration which results in protracted cases, because of the inability to retain and absorb fluid, due to the persistent vomiting. These infants become much depleted and shrunken and present a pitiable appearance, with shrunken fontanelles, feeble slow pulse, irregular breathing and evidence of acidosis. The urine is highly colored and scant, and in cases of anuria a mistaken diagnosis of renal disease has been made. I have been impressed by the fact that frequently these cases develop a fever rising to 102° F. or more. I agree with Grulee and Bonar,[11] who find an apparent direct connection between the reduced water contents of the body and the occurrence of fever in infants suffering from repeated vomiting. With increased intake of liquid food the temperature falls, while a rise of temperature results after thickened food. On this account they question the use of thickened food in the medical treatment of pyloris stenosis. Personally I have been in the habit of relieving the dehydration in these very sick, depleted infants by the administration, whenever possible, of hypodermoclysis, enteroclysis or, preferably, intraperitoneal injections of normal

saline solution. This often improves the prognosis of operation in otherwise moribund cases. The method of Helmholz—the transfusion of glucose solution through the superior longitudinal sinus—is said to be efficacious for a similar reason.

Treatment

Formerly, treatment of the condition was almost exclusively medical, because of the high operative mortality; but with the improved surgical methods within the last decade the surgical mortality has gradually declined, so that now all but the mildest cases are allowed only a brief period of medical treatment. It must be remembered, however, that the actual tumor of the pyloric muscularis present in true hypertrophic stenosis does not vanish under medical treatment. Downes concludes that if the patient is observed from the onset of symptoms, medical treatment may be tried for a period of not longer than ten days, provided the weight loss does not exceed 20 per cent during this time. If then the child does not show definite improvement, operative interference is indicated; also, patients under medical care who suffer a relapse, and finally, all cases with a history of ten days or longer, for whom the data as to previous weight are lacking, and in which the patient is not in very good condition, should be classed as surgical. The mortality among patients coming to operation within four weeks from onset of symptoms is less than 8 per cent.

The chief medical risk is acute inanition or, in less severe types, slow marasmus, the patient suffering from painful emaciation for several months. Final recovery is doubtful and the prospects from ultimate surgical intervention hopeless. Medical treatment is most successful in cases where the organic obstruction is not severe and the pyloric spasm is chiefly responsible for the symptoms. Theoretically, the spasm will gradually subside if nutrition can be maintained, but convalescence is always protracted and relapse not uncommon.

The treatment consists of careful feeding, stomach irrigation

and medication. The child should not be weaned, but the meal should be small and not too frequent. In bottle-fed infants the fat content should be low; skimmed milk formulas are said to give the best success. Some good results have been reported from the use of thick foods, such as farina and 10 per cent rice flour, as used in the idiopathic vomiting of infancy.[12] Gastric lavage with 1 per cent solution of sodium bicarbonate about 112° F. is practiced twice a day to rid the stomach of accumulated food and mucus, and seems to have some effect in allaying spasm. If the infant is much prostrated and dried up, the administration of normal salt solution, as previously mentioned, is indicated, Rectal feedings are of little value. Drugs, as a rule, are of no use, but the opiates, bromides or belladonna may be tried for their sedative and antispasmodic effects. It is most important not to wait too long before resorting to surgical treatment. The regrets, according to Griffith and others, are for undue delay in advising operation, but never for recommending early interference. The immediate performance of the Rammstedt operation offers a better chance of life than any other method of treatment yet proposed (Holt.)

Surgical Treatment

Previous to the last ten or twelve years the mortality of cases treated either by medical or surgical means averaged about 50 per cent. (In 1918 Dufour and Fredet collected 135 operative cases with a general mortality of 50 per cent.)

Formerly posterior gastroenterostomy in experienced hands gave good results. The combined statistics of Scudder,[13] Stillman,[14] and Richter,[15] representing 46 such cases, showed an operative mortality of 16 per cent, but the mortality with other operators, including Downes, was about 35 per cent.

The difficulty of a gastroenterostomy upon a baby but a few days old, with a tiny stomach and still tinier jejunum, not much thicker than a leadpencil, can be appreciated. Therefore, the operation of extra-mucous pyloroplasty by the Rammstedt

method (or Strauss' modification) has rapidly gained favor since its introduction in America by Downes. The operation can be performed in from fifteen to thirty minutes, the mortality being about one-half of that of gastroenterostomy. Downes[3] reports a total of 175 cases with 30 deaths, or 17.1 per cent mortality. Strauss[4], using his own method of pyloroplasty, reports 107 cases with 3 deaths, a mortality of 2.8 per cent. Even better results will no doubt follow when diagnosis is always made early and operation not delayed, and as the technic improves.

The credit for originating the operation of "submucous pyloroplasty," in reality belongs to the Frenchman, Fredet (1918). His operation consisted of a longitudinal incision through the serous and muscular coat of the pylorus, liberating but not opening the mucous layer, and then converting the longitudinal incision into a transverse one by sutures. Of his first 3 cases 1 died and 2 were successful.[15]

In 1910, Weber[17] recommended the same procedure, which he believed original on his part, but added a row of Lembert sutures, and called the operation "partial pyloroplasty," claiming it to be superior to the Loreta method (dilatation of the pyloric lumen from within by sounds introduced through a small gastric incision), and to the Nicoll method (essentially the excision of a V-shaped portion of the tumor and resuture of the oblique incision).

In 1912, Rammstedt, of Munster,[18] reported 2 cases following his new procedure. He used Fredet's operation of the longitudinal incision down to the mucosa, but permitted the pyloric wound to remain gaping. Inasmuch as the Rammstedt operation thus embraces Fredet's original idea, Downes justly designates it the "Fredet-Rammstedt operation." This method has become the method of choice in the treatment of hypertrophic stenosis of the pylorus. Not only does it lessen shock but it is much simpler, quicker and requires less surgical skill than gastroenterostomy. It preserves the continuity of the alimentary tract and permanently removes the obstruction, as shown by *x*-ray and postoperative evidence.

Preoperative treatment consists in emptying the stomach of food, gas and mucus by means of lavage. Hypodermoclysis immediately before operation is not advisable, since it aggravates the tendency to bleeding, especially in older infants. Most surgeons perform the operation under ether, but Bevan advocates local anesthesia in every case. One-tenth per cent novocaine may be used.

A right rectus incision about one or one and one-half inches long should be made, opening the abdomen. Incisions in the linea alba are liable to break open after operation or to injure the intestines, owing to the thinness of the abdominal wall in this region. After bringing the pylorus up into the wound, the thickened pyloric muscle is divided in its whole length and thickness from the stomach to the duodenum by a longitudinal incision, avoiding the small bloodvessels beneath the peritoneal covering. As the mucosa is approached the remaining muscle fibers are spread apart by blunt dissection with the handle of the scalpel. This avoids nicking the mucous layer, a serious accident. Particular care is necessary at the duodenal end of the tumor, where the change from the thick-walled pylorus to the normal duodenum with its thin muscular layer is quite sudden. In case an opening is made in the mucous membrane it should be immediately closed with small hemostat and ligature. Following the pyloric incision, as its sides are separated, the mucous membrane bulges into the wound, the lumen of the pylorus is accordingly enlarged and the obstruction relieved. The pylorus is then dropped back into the abdomen, but not until all bleeding has stopped. Hot pads or fine ligatures may be required for excessive bleeding. It is not necessary to cover the pyloric incision with omentum. The abdominal incision is then closed, always with through-and-through silkworm-gut sutures. In addition the various layers may, if practicable, be restored.

Strauss,[19] of Chicago, has devised a modification of the Rammstedt operation, the advantages of which he claims are lower mortality, greater patency of the pyloric lumen, very little postoperative vomiting, and shorter convalescence. He makes the

usual Rammstedt incision and spreads apart the cut edge of the pyloric sphincter. With the handle of the scalpel, he frees the circumference of the mucosa from the muscularis, unfolding the infolded mucosa. A muscle flap, prepared from one of the sides of the incision, is then brought over to cover the gaping incision and is sutured, thus providing a larger pyloric passage entirely surrounded by muscle. He states that the shelling out of the mucosa at once establishes a free passage for the food. The site of operation is covered with an edge of omentum, which is sutured in place.

Postoperative Care

Body heat is maintained and stimulants given if necessary. Feeding may be begun almost immediately after the child comes out of ether. Breast milk in small quantities is desirable or, if the baby is bottle-fed, a low-fat formula, one or two teaspoonfuls every two hours, with a dram of boiled water between feedings. The amount is gradually increased, so that in two or three days the child is receiving an ounce of milk or mixture every three hours, with water in the intervals. Breast-feedings also are at first limited in amount, regaining the normal in about one week, after careful supervision.

If, following the operation, sufficient liquids are not taken, proctoclysis or hypodermoclysis of normal saline or sodium bicarbonate solution will be useful for a few days. The early postoperative vomiting can usually be relieved by a semi-erect posture with gastric lavage. If bowel movements have not set in after twenty-four hours, a dram or one and one-half drams of castor oil should be given.

Convalescence is soon noted by the gradually increasing weight and the appearance of normal stools; most cases are discharged within twenty days in an entirely satisfactory condition.

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DISCUSSION ON THE PAPERS OF DRS. VANDER VEER AND HARRY C. DEAVER

Dr. John H. Jopson, Philadelphia:

Dr. Vander Veer's paper recalls to my mind a case of rather unusual complication after gastroenterostomy in my own experience. This patient, a man, had been operated upon by me thirteen years before for an acute perforation of a pyloric ulcer. He had had ulcer symptoms for some time previously. He consulted a well-known internist, who took a test meal and made sundry other examinations. On the way home from the doctor's office the ulcer ruptured. Operation the same evening with suture of the perforation was followed by a good recovery. Two years later, in response to a recurrence of the ulcer symptoms and pyloric obstruction, I did a posterior gastroenterostomy, with prompt relief. Nine years later I saw him with an attack of acute intestinal obstruction. At operation I found, just below the site of the gastroenterostomy opening, which was functioning perfectly, a band of adhesions stretching across the jejunum from a neighboring portion of the mesentery and occluding the gut. Removal of the obstruction was followed by rapid recovery.

In regard to the jejunal ulcers I was informed recently by one of the internists at the Mayo Clinic, who had made a special study of the subject, that in practically every case in which they had occurred there was a history of some postoperative complication, indicative of a fault in technic.

In reference to Dr. Deaver's paper, we are seeing more and more of these cases at the Children's Hospital. In a small series of my own, treated by the Rammstedt method, the mortality has been the same as mentioned by Dr. Deaver, namely 8½ per cent.

Fortunately most of the cases I have been called to operate upon have been turned over to me in good condition by Dr. J. P. Crozer Griffith and Dr. Alfred Hand, pediatrists of wide experience, and operation in this early and favorable period is followed by a low mortality.

It has seemed to me that some symptoms which are supposed to be typical and indicative of stenosis were lacking. I have seen a number of infants in whom no tumor was palpable before operation, and this because the pylorus was tucked away high up and far back under the liver. In two or three instances the cases have been operated on under a mistaken diagnosis and no tumor was found. I do not think this is a great reproach, because these cases can be opened

and closed, if nothing is found, in five or six minutes. The cases of this kind which I have encountered all went along smoothly to recovery. The physicians were able to give their undivided attention to the feeding problem, and they seemed to think they were rather benefited than otherwise by the operation. I can see no reason for modifying the Rammstedt operation. I think it is ideal. My own practice has been to outline with the scalpel the extent of the incision on the pylorus by a rather superficial division of the circular muscular fibers, and then beginning on the stomach side I have practised blunt division of the muscle down to the mucosa by tearing the deeper fibers apart with the Mayo scissors rather than by dividing them with the sharp edge of the knife. In this way I avoid doing any damage to the mucosa. The chief anxiety in my mind has not been the operation itself but the anesthesia.

I have had the opportunity to perform one secondary operation, and the findings were rather interesting. This case had been operated upon primarily by one of my colleagues. The vomiting recurred after a time, and some weeks after the first operation I reopened the abdomen. At the site of the pyloric incision there was a little gap in the muscle, which was covered by a thin layer of scar tissue, much as we see a small gap in the scar after a laparotomy, where the fascia is not thoroughly approximated. I divided the circular sphincter fibers more extensively on the stomach side of the pylorus in this case, and the recovery was complete and permanent.

Dr. Dallas B. Phemister, Chicago:

A complication of posterior gastroenterostomy that I have seen four times at autopsy is jejunal ulcer with adhesions to and perforation of the transverse colon. If the ulcer forms about the anterior portion of the gastroenterostomy it may easily lead to adhesions between the jejunum and the overlying transverse colon, and if perforation and fistula formation occur marked symptoms result. The symptoms may be gastric, from the escape of large intestine contents into the stomach, and they may be colic, from the passage of gastric contents directly into the colon. In the four autopsies which I saw death had resulted two and a half months, two years, eight years and ten years after performance of the gastroenterostomy. In the two earlier cases the ulcers were still active, but in the older cases they had healed, leaving the colic fistula. In the oldest case the ulcer formed at the right side of the gastroenterostomy opening and produced marked stenosis of the jejunum with enormous dilatation of the duodenum and jejunum proximal to the obstruction.

Dr. John B. Deaver, Philadelphia:

I should like to ask Dr. Vander Veer whether these two cases were both marginal ulcers? Marginal ulcer holds about the same relationship to gastroenterostomy as do adhesions after abdominal section. Both are more or less the bugbears of surgery.

As to the etiology of these secondary ulcers, I believe the factor is the same as in the primary ulcer, and I think it is generally conceded that this is infection. Of the many cases of ulcer that go to perforation I believe that the appendix is the original focal infection. I believe also that we should look carefully for other sites of focal infection, particularly the gall-bladder. It is not uncommon for me to remove the gall-bladder in ulcer cases, and I never leave an appendix in if it is present.

About 2 per cent of all my gastroenterostomies have been followed by marginal ulcer. I may say that I am now much more radical in my gastric surgery than formerly. I never leave an ulcer if I can possibly excise it. Where this is not possible I do a pylorectomy, or a subtotal gastrectomy, or a circular resection. I believe in future I shall see fewer marginal ulcers.

There seems to be an idiosyncrasy on the part of some patients to form ulcers. In one case I have operated three times for marginal ulcer. I have removed nearly all of the stomach and the patient still returns with symptoms. I have concluded to let him alone. These are trying cases. The unfortunate thing is that these marginal ulcer cases often get into the hands of the gastrologist instead of coming back to the surgeon. When a patient comes to me complaining of postoperative symptoms similar to those before operation, without examining him I say: "You had better go to the hospital: you have a marginal ulcer." The *x*-ray plate, while it does not always show these marginal ulcers, does indicate the presence of obstruction sufficient to justify operation.

One point with regard to marginal ulcer may be of interest. I believe it is important to make the opening in the mesocolon nearer the vertebra than the colon. The farther you keep away from the colon in the presence of marginal ulcer the less likelihood will there be of infiltration of the colon and the less likelihood of a fistulous communication with the bowel.

Dr. Miles F. Porter, Fort Wayne, Ind.:

I should like to ask Dr. Deaver if he makes a large opening?

Dr. Deaver:

I make a small opening, only about one and a half inches.

Dr. William A. Downes, New York:

We have operated upon something over 250 cases of pyloric stenosis in infants by the Fredet-Rammstedt method, and the mortality has varied in different years from 10 to 20 per cent according to the condition of the babies. In the cases that come within a week or ten days after the onset of symptoms the mortality is under 2 per cent.

I have recently had a reprint from Fredet, the originator of the extramucous pyloroplasty, in which he states that he has given up this type of operation and gone back to gastroenterostomy. The results obtained in either type of operation do not compare favorably with those obtained by several surgeons in this country. Personally, I believe that a palpable tumor at the pylorus is the most important diagnostic sign. If care is used in emptying the stomach of gas by passing a tube and relaxing the child by giving a bottle in practically every case you will be able to feel the thickened pylorus on gentle palpation.

As to anesthesia, a year ago Dr. Bevan suggested that I try local anesthesia, and since then we have done fifteen cases under novocain, selecting those in poor condition. The results have been satisfactory, and this form of anesthesia is now routine at the Babies' Hospital in these cases if at all depressed.

As to opening the duodenum I have had this accident occur three times. It happened early in the series, and in each instance the result of using a knife to divide the muscle. Only the superficial portion of the muscle should be divided with the knife and the separation completed with small blunt-pointed artery forceps. The results following the Fredet-Rammstedt operation have been remarkable. These babies seem to be in better condition and gain weight more rapidly than otherwise normal babies.

In reference to gastrocolonic fistula I have had two cases. Both came to operation and made satisfactory recoveries.

Dr. Frank E. Bunts, Cleveland, Ohio:

The case which Dr. Jopson related recalls to my mind a very interesting one of a similar character that occurred this winter. In the early days the first case I had was an anterior gastroenterostomy with recovery, and this encouraged me to go on with the operation a good many years ago. Then followed the posterior operation, and, until I adopted the Rammstedt method, I began to feel that the operation was not a success. Last winter I operated on a child, doing the Rammstedt operation. The baby did well for a week, when the symptoms all recurred and it looked like a failure. I opened it up and intended to

do a posterior gastroenterostomy, but on examining the site of the operation I found that the tumor looked as it did at the first operation. It had healed completely. There was no dilatation or thinning there, and I knew that I had made an incomplete operation. I then made a second incision parallel to the original one, doing a more complete operation, which was entirely satisfactory.

Dr. Arthur W. Elting, Albany, N. Y.:

I should like to report an unusual instance, the occurrence of three cases of congenital stenosis of the pylorus in the three and only children of one family. The first case occurred ten years ago. When the child was brought to the hospital it was almost moribund. A posterior gastroenterostomy was done. The child died. The second child a few years later developed the same condition, but because of the experience with the first case the parents declined operation and the child died. A few months ago the third child in this family, then a few weeks old, was brought to the hospital with a typical congenital stenosis of the pylorus, and a Rammstedt operation resulted in a speedy cure.

I recall another family in which two successive children had congenital stenosis of the pylorus. The first one I operated upon and the second was operated upon by Dr. Downes.

Dr. John C. Oliver, Cincinnati, Ohio:

X-ray pictures taken in two of my cases in which gastroenterostomy had been done showed that after the lapse of thirteen and fourteen years after operation all of the barium passed through the gastroenterostomy opening. This certainly proves that the pyloric obstruction remains permanently.

Dr. Harry C. Deaver, Philadelphia (in closing):

In my series of cases, which has been seventy in number, I have been able to palpate the tumor in about 60 per cent, but the size of the tumor does not have much bearing on the amount of obstruction. My experience has been that very often one has more obstruction with the smaller hypertrophied pylorus than he does where the hypertrophy is much larger.

As to the mode of procedure, I cut well beyond the margin of the hypertrophy and through the hypertrophied muscle. As I approach the mucous coat I use a blunt dissector, gradually exposing it. In this way I have avoided opening the lumen of the pylorus.

The late cases that come to us, those which are practically moribund, I find that many of them are suffering with anuria. Hypodermoclysis has been used without much benefit. Lately I have been injecting saline into the peritoneal cavity. It is remarkable how well these children respond, and in thirty-six to forty-eight hours one can operate, and they make good recoveries. It is the late cases from which we get a high mortality. If the cases are recognized early and operated the mortality is practically *nil*.

CONGENITAL ABSENCE OF THE LOWER PART OF THE ILIAC MESENTERY.

By GEORGE E. ARMSTRONG, M.D.
MONTREAL, CANADA

By one of those strange coincidences that we are all familiar with the two patients presenting the rare anomaly that I now report were in the Royal Victoria Hospital at the same time. The anomaly consists in the absence of a mesentery for the terminal six or seven inches of the ileum.

My own case was observed in a man, aged thirty-nine years, sent up to me as a case of recurring appendicitis. He gave a history of having had three attacks at intervals of a month or six weeks. The outstanding feature in his history was the severity of the pain during the attacks. He told us that during his last attack, from which he was just recovering, morphin failed to give him relief and that he had been kept under the influence of ether from 11 P.M. until 6 A.M. the following day. He referred all his trouble to the region of the appendix, and on admission the right lower quadrant was definitely tender. Seventy-two hours after admission I proceeded to uncover the region of the appendix. The condition observed on opening the abdomen was most unusual. The cecum was very mobile and on pushing the cecum and ascending colon inward the tissues outside the colon were found to be hemorrhagic, ecchymosed and at one spot the peritoneum was found lacerated. The appendix was curled up external to the ascending colon and retroperitoneal. It was congested. The area external to the

colon looked as if it had been kicked with a heavy boot. The terminal six inches of the ileum were subperitoneal. With the cecum in its normal position it ran a course forming a loop with rather a sharp turn or kink at its point of juncture with the posterior aspect of the cecum. This whole loop when the cecum was first lifted up was completely flattened and so not noticeable. Manipulation of the small gut and cecum caused the lower end of the ileum to fill with gas and so come into view. It is probable that the anomaly caused a partial obstruction under certain favorable conditions.

The deficiency in the mesentery began in the pelvis in front of the sacroiliac synchondrosis about one inch below the brim of the pelvis. From this point the ileum passed up over the external iliac vein and over the psoas muscle to its junction with the cecum. It was very intimately adherent to the wall of the external iliac vein by normal tissue. There was no sign of inflammatory action.

The appendix was removed and the cecum and ascending colon were sutured to the outer abdominal wall by three rows of sutures. So far there has not been any recurrence of symptoms.

The other was discovered at autopsy. The patient had been under the care of one of my colleagues. On tracing the small gut downward it was found to be free from adhesions or exudate and apparently ending suddenly at the brim of the pelvis over the right sacroiliac joint. On lifting up the cecum, which was markedly dilated, it was found to be very mobile and had a definite mesocolon and apparently disconnected from the ileum. This was due to an anomalous condition of the last six inches of the ileum. The mesentery ended abruptly at the brim of the pelvis over the right sacroiliac joint, where the ileum became retroperitoneal and ran posteriorly across the iliopsoas muscle and then turned upward and for nearly three inches lay in a groove between the iliopsoas and quadratus lumborum. It then reached the posterior surface of the cecum, on which it ran downward.

The only similar cases that I have found recorded in medical literature were reported in May, 1890, by W. H. Bennet, Surgeon to and Lecturer on Anatomy at St. George's Hospital, and Rolleston, Curator of the Museum.[1] They reported three examples of the anomaly, their attention being called to the condition by the fact that it was "associated with and probably instrumental in producing a fatal twisting of the lower part of the ileum."

The cases that were observed in the Royal Victoria Hospital differed from those reported by Bennet and Rolleston in the length and mobility of the cecum and ascending colon.

In their cases the cecum had not descended into the right iliac fossa and lay over the right kidney. Moreover the cecum was small and of the fetal type.

It is interesting to note that since these cases were observed Prof. Whitnal (anatomy) and Prof. Simpson (histology and embryology) have found two similar conditions in fifty bodies in the dissecting room of McGill University. In the latter there was no clinical history and no evidence that the anomaly had contributed to the death.

Normally the ascending mesocolon of the fetus is flattened against the posterior abdominal wall on the right side and fuses with the parietal peritoneum over an area shown in Diagram A.

Over this area the posterior layer of the fetal mesocolon and the fetal parietal peritoneum are obliterated and the anterior layer of the fetal mesocolon becomes the parietal peritoneum of the adult. This is shown in Diagrams B and C.

Normally this fusion of the ascending mesocolon begins at the level of the right colic flexure and extends downward.

In the cases that I report this fusion has apparently extended downward to include the mesentery of the terminal part of the ileum as shown in Diagram D.

A very possible cause of this extension of the area of fusion is the presence in the fetal stage of a "genitomesenteric fold"

[1] Jour. Anat. and Physiol., vol. xxv.

(see diagram), which by putting traction on the developing mesentery may cause a fusion of the ileum to the posterior abdominal well.

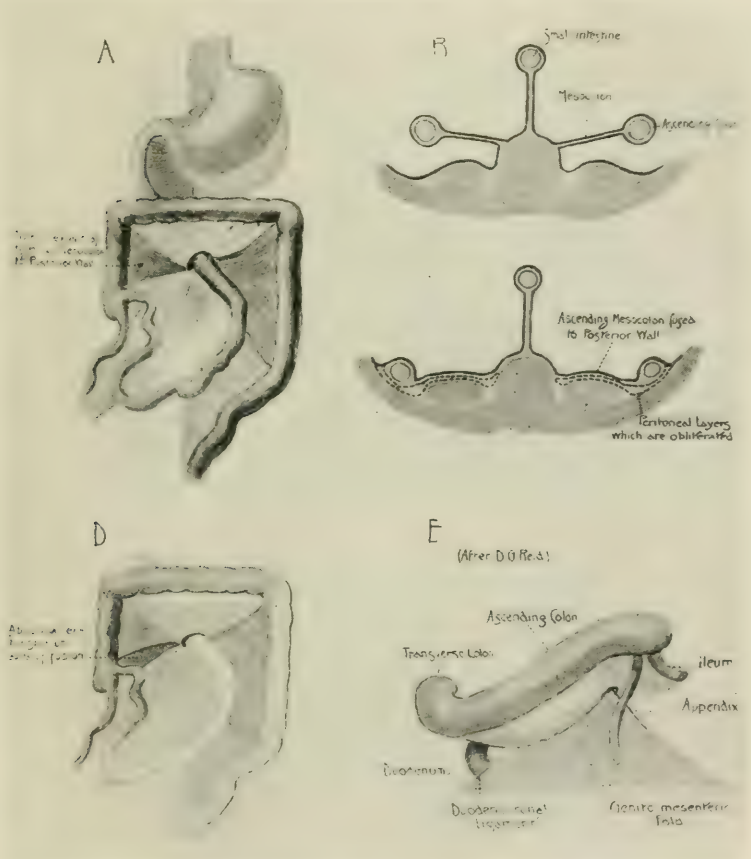

FIG. 1

Douglas G. Reid in "Studies of the Intestine and Peritoneum in the Human Fetus,"[1] describes a "genitomesenteric fold" found in eleven of twenty fetuses examined.

[1] Jour. Anat. Physiol., 1911, xlv, 74–84.

It passes from the inferior surface of the mesentery in the right half of the abdomen, forming an anteroposterior septum, one lying in the vertical plane and incompletely dividing the portion of the abdominal cavity below the root of the mesentery

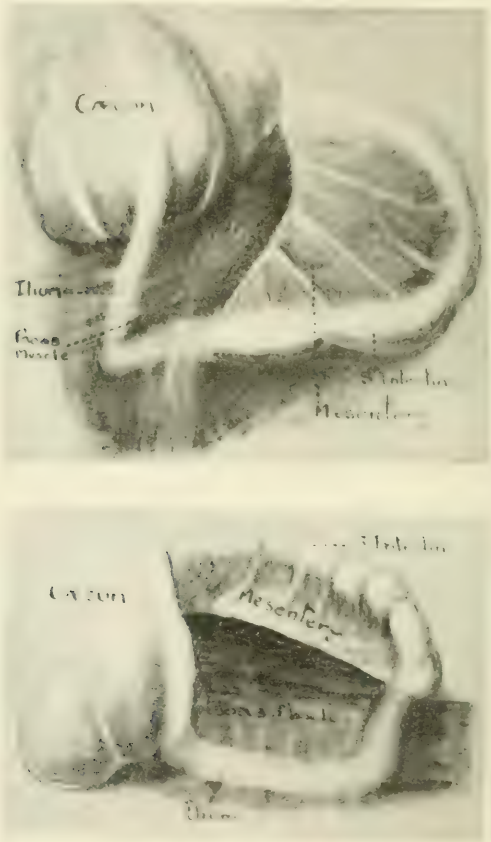

FIG. 2

into two compartments. The smaller of these is on the right side and contains the cecum, the larger, on the left, contains the pelvic colon.

The fold is triangular and has two attached borders (superior and posterior) and one free (inferior). The posterior border is

attached to the posterior abdominal wall along the line of the right spermatic or ovarian vessels. The superior, or mesenteric, border is attached to the inferior surface of the mesentery often to a considerable extent. This attachment to the mesentery is generally exactly along the line of the iliac branch of the ileocolic artery.

The upper part of the fold may persist as the free edge of the duodenorenal ligament of Huschke. Its lower part may persist in the adult, forming the medial boundary of the retrocolic fossa, or passing from the ileum or the appendix. It is the commonest cause of a retrocolic position of the appendix.

"The fold may also assist in producing adhesions of the duodenum, cecum and ileum to the posterior abdominal wall. Adhesion in the adult binding down the terminal part of the ileum—the cecum, the appendix, the mesentery, the mesoappendix and even the bloodless fold of Treves—need not be the result of pathologic changes unless the natural causes we have indicated for the adhesion can be excluded, although a genitomesenteric fold may not always be seen even in the fetus." (Page 82.)

EXTRAPLEURAL RESECTION AND PLASTIC OF THE THORACIC ESOPHAGUS. AN ORIGINAL METHOD. REPORT OF A SUCCESSFUL CASE WITHOUT GASTROSTOMY

By HOWARD LILIENTHAL, M.D.
NEW YORK

For many years cancer of the thoracic esophagus had been the despair of surgery. Then, in 1913, came the brilliant achievement of Franz Torek, of New York, who relieved a woman over sixty years of age by boldly invading the posterior mediastinum through the pleura and, in the face of discouraging obstacles, extirpating the tumor-bearing gullet. His patient is alive today, swallowing her food through a tube of rubber which completes the passage between an esophageal opening in the upper thorax and a gastrostomy. In spite of the perhaps trivial annoyance of keeping two wounds clean, the state of this patient is by no means melancholy, and the success of the operation has been the cause of perhaps hundreds of attempts not only to repeat the feat of Torek, but to place transpleural thoracic resection of the esophagus among the justifiable operations of surgery. But unfortunately neither Torek himself nor any of his followers has been able to save another of these patients, while hundreds of failures have resulted. So surely did the fatality follow the operation that some conservative surgeons believed that Torek's success was far from being a benefit to humanity.

Dr. Bevan, speaking at the 1915 meeting of the American Surgical Association, appeared to doubt the wisdom of continuing

the operative attack upon the thoracic part of the esophagus. For some years radium and the *x*-rays have taken the place of the knife with what seems to me a less merciful ending; for the last day was merely put off with false hopes, longer suffering and greater expense to the patient or his friends.

Probably the greatest danger in esophageal surgery has been infection of the mediastinum, whether the avenue of approach has been through the pleura or outside of it, and many experiments have been made to avoid this great peril. At the end of this paper is a list compiled for me by Dr. S. Hirshfeld, of New York. It by no means gives all the references in the literature, but it covers the various sides of the subject pretty well.

The only successful cases recorded are those in which some form of esophagoplasty or anastomosis has been made for benign stricture without resection. Much ingenuity has been shown in devising tubes of skin or hollow viscera, all placed extrathoracically, to sidetrack the impervious part of the gullet, even the stomach itself having been drawn out of the abdomen and transplanted to take the place of a part of the esophagus.

About four years ago I began to give serious thought to the problem and it occurred to me that in order to avoid mediastinal infection it would be necessary to divide the procedure into at least two parts: (1) the exploratory isolation of the esophagus with obliteration of the mediastinum, and, (2) the resection and plastic. I hoped to enter the chest from behind and without invading the pleural cavity. Different levels of the esophagus might demand various modifications of the method selected, but I finally decided to try out the possibilities with growths occupying the esophagus below the aortic arch.

I was by no means sure that enough of the pleura could be stripped free to make safe the work on the esophagus. I hoped that even if the pleura were to be injured the method itself need not be abandoned. It would simply mean that the interval between the isolation of the esophagus and its resection would have to be sufficiently long for the pleural infection to have disappeared. Through the kindness of Dr. J. McAllister I was

enabled to test out the method on a cadaver in the morgue of Bellevue Hospital. Dr. Harold Neuhof, associate surgeon to Mt. Sinai Hospital, assisted me in this investigation as well as in the operative work on living subjects. I am glad to acknowledge here his sympathy and his resourcefulness, which did much to smooth the way in solving this difficult clinical problem. We found, as I feared, that the pleura stripped with difficulty in the subject upon which we worked, but we believed that another pleura might prove more resistant to handling.

The question came up as to whether the approach should be from the left or the right of the spine, and from a fairly large experience in thoracic surgery I concluded that the left approach, although no nearer the esophagus than the right, would probably prove simpler and safer: (1) because the descending aorta would form the first important landmark; (2) because we would be further away from the important veins and the thoracic duct; and, (3) because on working on the right side it would be difficult and perhaps impossible to free the pleural fold, which crosses the median line in this part of the thorax. Indeed, in doing a left transpleural operation upon the esophagus I had once entered the right pleura in loosening the gullet from its bed. The demonstration on the cadaver proved conclusively that it was quite feasible to reach the esophagus through a sufficiently large wound without the resection of many long pieces of rib. It could probably be done even without costectomy, merely dividing several ribs close to the spine, cutting the intercostal structures down to the pleura, then, after peeling off the pleura, gradually spreading the wound. We finally concluded that the subperiosteal removal of six or seven inches of one rib would facilitate the pleural stripping and subsequent exposure of the mediastinum. I realized that it would probably be necessary to transplant skin by grafting of some sort in order to prevent cicatricial closure of the space between the severed ends of the esophagus. How this was finally accomplished, the history of my case and the illustrations will explain.

CASE.—Davis F., aged thirty-four years, entered Mt. Sinai Hospital on December 14, 1920. For a year there had been cough with slight expectoration. There was some nasal obstruction which caused him to be a mouth-breather. About nine months before admission he began to suffer pain behind the sternum from the neck to the epigastrium on swallowing solids. For three months he was obliged to wash down his meals with draughts of fluid on account of the presence of an obstruction. I am indebted to Dr. Sidney Yankauer for referring this patient to me; Dr. Yankauer having removed through the esophagoscope a specimen which was pronounced by Dr. Mandlebaum to be carcinoma of the squamous cell type.

The Wassermann blood examination was reported as 4+. Further examination by Dr. Yankauer failed to show the full extent of the tumor because the obstruction left a passage too small for his instruments to pass. The general physical examination showed nothing remarkable. The urine analysis was negative. The blood was apparently normal and belonged to Group II. There had, of course, been loss of weight, but the general nutrition had not greatly suffered. The *x*-ray films brought by the patient when he entered the hospital were not satisfactory except to demonstrate an obstruction, which could be seen close to the dome of the diaphragm, and a narrow streak of barium running downward from the supposed location of the tumor, through the shadow of the diaphragm until it was lost below. Fluoroscopically there was partial obstruction, which was interpreted by the men of the *x*-ray staff as being just above the cardia. It is best in these cases not to depend upon the fluoroscope alone, but to have good plates or films made taking in the entire chest so that the level of the tumor may be designated by the corresponding vertebra.

With a positive diagnosis of carcinoma of the esophagus, probably low down and reported by the *x*-ray department to be "two inches above the cardia," operation was undertaken December 16, 1920. The anesthetic, nitrous oxide ether, was administered by Dr. Branower, Dr. Neuhof acting as first

assistant and Dr. J. Lawrence Jones, house surgeon, as second assistant.[1] An intravenous injection of 6 grams of sodium citrate in solution was administered by Dr. Hirshfeld to reduce the coagulating time of the blood.[2]

Operation. The patient was placed on his right side, with his knees and hips flexed and the trunk bowed forward. The bridge of the table was raised so as to cause a scoliosis with the convexity toward the left. The incision was made from a point about one and a half inches to the left of the spine, along the eighth interspace to the midaxillary line, then downward and backward along the tenth rib, outlining a skin flap with its pedicle posterior. This flap was now dissected up, together with the subcutaneous fat and here and there a bit of fascia and muscle, and turned back and wrapped in warm, moist towels. (See Fig. 1). Five excellent drawings were finished from sketches made at the operation. The artist is Dr. J. B. Stenbuck, senior on the house staff of Mt. Sinai Hospital. Through the resulting wound area another incision was made upon the ninth rib, the greater part of which was subperiosteally resected. Beginning toward the back, the pleura was dissected with the finger away from the posterior chest wall, and then forward from under the surface of the tenth rib, which was divided posteriorly close to its angle after it had been freed from the underlying pleura. With the pleura peeled away and the lung pushed forward with it the structures in the posterior portion of the thorax were exposed and easily recognized. (Figs. 2 and 3.) The first important landmark was the great splanchnic nerve, and the next the descending aorta, from which the pleura separated easily. A stomach tube was passed through the mouth into the esophagus, down to and finally through a resistance (at the level of the tumor) which had been quite easily overcome, then on into the stomach. The

[1] I desire to acknowledge the conscientious attention, also, of the surgeons of my house staff, Dr. L. Mason Lyons having taken the place of Dr. Jones soon after the operation.

[2] A method of Dr. Harold Neuhof. See paper by Neuhof and Hirshfeld N. Y. Med. Jour. January 15, 1921, vol. 113, No. 3, p. 95.

esophagus below the tumor was thus rendered palpable, as it lay anterior to the aorta, drawn forward by its connection with the pleura much as the ureter is drawn forward with the peritoneum. No tumor was felt as far down as the opening through the diaphragm, which by the way was exposed with ease and the open-

FIG. 1.—The skin flap dissected off and the beginning of the incision in the denuded area.

ing explored with the finger. A portion of the eighth rib was now freed from the pleura and a short resection done. Then the seventh and sixth ribs were divided posteriorly, also extrapleurally, and the rib separator introduced, retracting the entire wound, especially the upper portion. The lower part could be

exposed even without the rib-spreader. Now the tumor could be distinctly felt as a fusiform enlargement of the esophagus about three inches below the arch of the aorta and extending upward to about one and a half inches below the arch. The

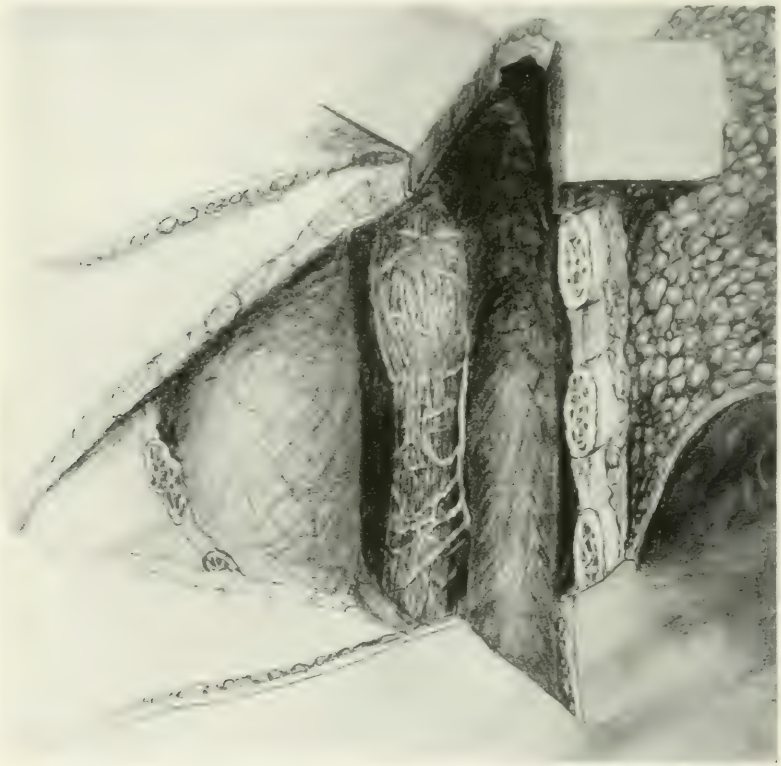

FIG. 2.—Esophagus exposure. Note lung covered by pleura, aorta, esophagus and (probably) the *right* vagus with its plexus gulæ.

esophagus was isolated and the right pneumogastric nerve freed from it, dividing the branches with scissors so that the nerve lay on the posterior wall of the chest. Following the advice of Torek this nerve had been anesthetized with 5 per cent cocaine solution on a pledget of cotton. The left vagus was not seen. It was probably separated bluntly from the esophagus during the free-

ing of this structure from the pleura. A thin rubber tube was now passed around the esophagus above the tumor and another below it, these tubes acting as suspension ligatures, so that the esophagus with its tumor was nicely exposed, making a beautiful demonstration for spectators. The skin flap was now placed in position, lying between the vagus and the gullet, its cuticular

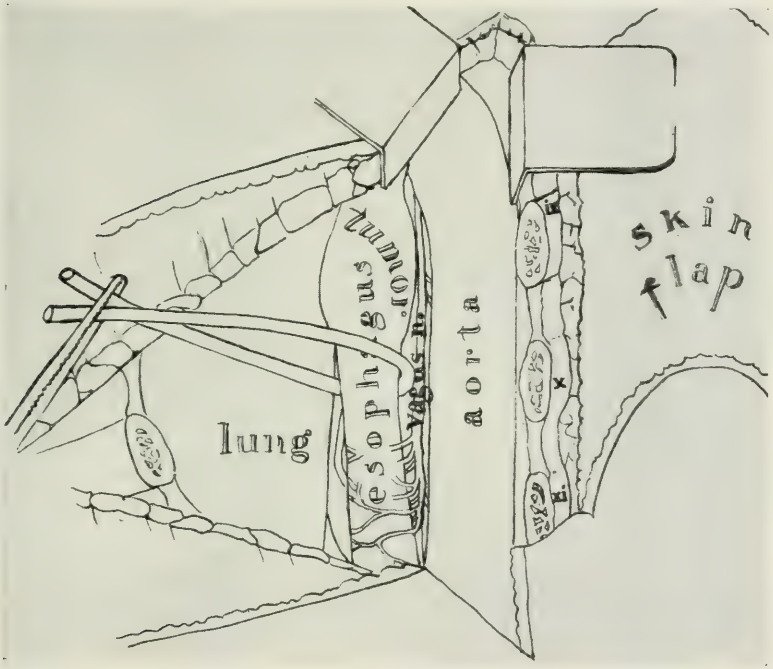

FIG. 3.—Diagrammatic, to explain in part Fig. 2. Note also the rubber tube placed around the esophagus to act as a retractor.

surface being next to the esophagus and its terminal portion being turned around the viscus so as to form a trough of skin to be completed into a tube by the division of the pedicle of the flap at another time. The skin flap was held in position by a suture of silk running from the tip of the flap to the inner part of the chest wall. (Fig. 4). Thus far the pleura had remained

intact, but now an additional portion of the eighth rib was resected and there occurred an accidental tear about one-half centimeter square. The lung, which had been sliding smoothly against the inner side of the pleura, changed its appearance the

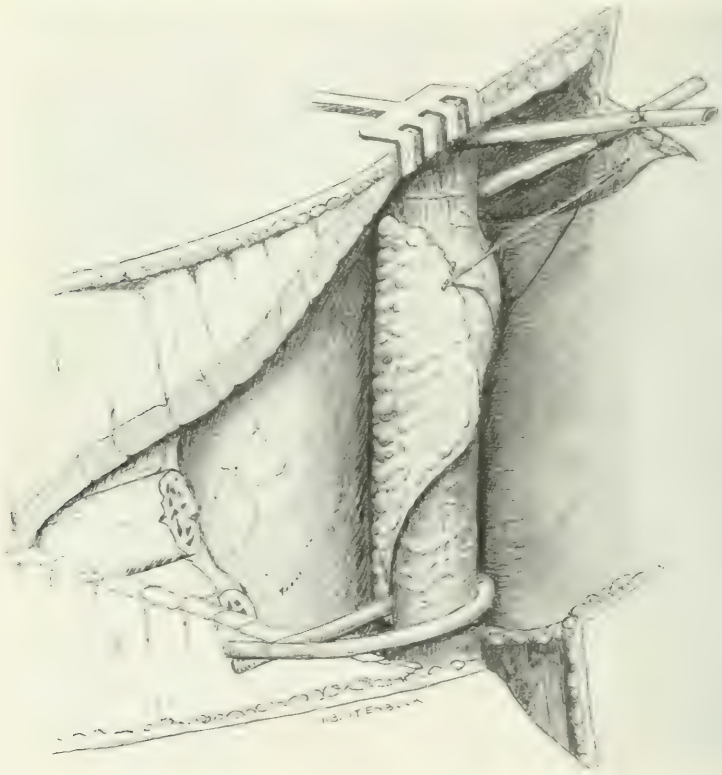

FIG. 4.—Skin flap partly surrounding esophagus and held in place by a suture to the chest wall. The two rubber tubes are left in place to act as retractors at the next step of the operation.

moment a little air entered, but the opening was quickly and completely closed by catgut sutures. Three heavy chromacized catgut stitches placed pericostally between the tenth and eighth ribs approximated what remained of these ribs, and the

anterior part of the wound was closed by suturing the divided muscles, the posterior portion remaining open.

Dressing. The pleura was covered with a large piece of rubber dam, extending down as far as the esophagus, which with its partially surrounding skin-flap lay at the bottom of the wound in its normal position. The remainder of the wound was lightly packed with gauze so as not to make pressure on the flap. No bandage or binder was used, the dressing being held in place by adhesive strips.

There was no shock following this extensive procedure. The patient lay comfortably on his right side. There was some mucus in the trachea, but the man was strong and easily able to expel it. He swallowed better than before the operation, possibly because of the dilation by the stomach tube. His highest temperature was about 102,° his pulse 112, but always strong.

My next note is December 23, one week after the operation. The gauze had been removed and replaced and the rubber dam had been taken out a day or two before. The tongue of skin-flap which surrounded the esophagus was slightly discolored, but only about one-half inch at the tip finally necrosed.

December 29, the wound looked well and granulation had begun. The cough had persisted, with about two ounces of expectoration in twenty-four hours. Later it completely disappeared.

December 29, thirteen days after the operation, swallowing became increasingly difficult, so it was decided to perform the second step without delay. A hypodermic of $\frac{1}{4}$ grain of morphine and $\frac{1}{150}$ grain of atropine was administered and the patient taken to the operating room. Dr. Neuhof and Dr. Ira Cohen assisted at this stage, and we were glad to have Dr. Torek present as a spectator. There was no anesthesia, either local or general, and the patient sat up across the table, bending forward, and making no complaint throughout. Granulating adhesions between the ends of the severed ribs were broken down with the fingers and large blunt retractors were used to expose the depths of the wound. Adhesions had taken place between the esophagus and the underlying structure and even between the

esophagus and the edges of the skin-flap. It was easy to find our way, however, because of the long rubber tubes which had been left around the esophagus at the close of the previous operation. The entire esophagus was covered with healthy granulations, but the tumor was still both palpable and visible in spite of this.

The esophagus was entered with scissors just above the tumor and an incision was made transversely below it. With one blade of the scissors in the lumen, the esophageal canal was opened in a sagittal direction, so that the finger could be inserted for exploration. The tumor limits were easily made out, the cross-section of the gullet above was completed, the diseased segment drawn with forceps out of the trough of skin, and the resection completed below. An immediate examination of the specimen showed that we were well beyond the limits of the growth, but to make doubly sure a small segment above this was cut away with the scissors. Hemorrhage was slight, no vessels being caught and no spurters seen. There was a little oozing in the upper portion of the wound. The skin-flap had already formed a trough and the mediastinum was sealed off by it and by granulation tissue. A "T" tube had been prepared, the cross-bar of which was to enter the upper and lower portions of the esophagus while the upright protruded from the wound for feeding. This proved impossible, however, because the injected fluid went in both directions and regurgitated from above. A swallow of water at once demonstrated that there would be escape alongside of the tube in the upper segment of the esophagus. The whole wound was lightly packed with gauze and the patient sent to bed in splendid condition. His color was good, his pulse strong, and there was a smile on his face. About one and a half hours later, however, it was found that he had bled considerably from a small artery in the upper segment of the esophagus. Retractors exposed the depths of the wound and the vessel was caught by Dr. Neuhof with a clamp which was left in place. Although the pulse had risen to 120 and the patient was pale and somewhat shocked from the loss of blood, his condition was not desperate.

A second dose of sodium citrate was given by Dr. Hirshfeld, subcutaneously this time, although all the bleeding had ceased since the vessel had been clamped. A few hours later the pulse-

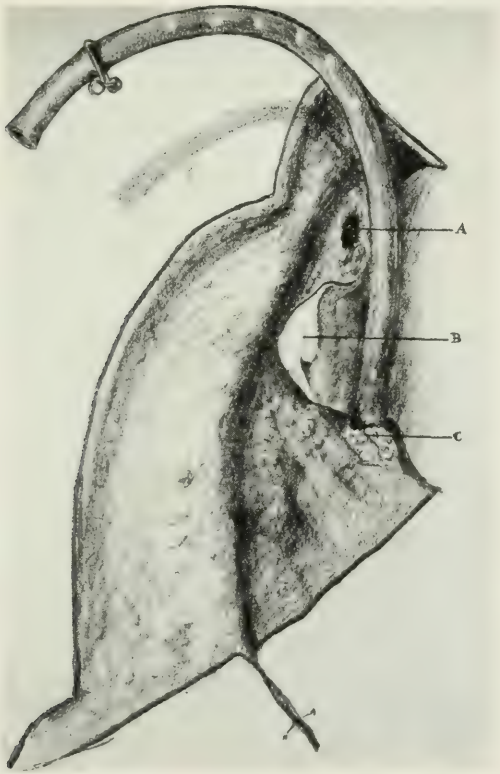

FIG. 5.—After the resection. Stomach tube in lower esophageal orifice and the skin flap upon which the rubber stomach tube rests. The white portion is the granulating side of the flap, not the epidermal side. The mediastinum is obliterated by granulation tissue, which also covers the other structures in the picture.

rate had receded to 116 and there was great improvement. Still not wishing to omit any precaution, I requested that a transfusion be performed, and this was done by Dr. Ottenberg, who injected 500 c.c. of blood by the citrate method. From that time on the

patient never gave us a moment's anxiety so far as danger to his life was concerned. Two days after the operation he was able to sit up during the dressing and the clamp came away without mishap. The "T" tube having proved useless was replaced by a stomach tube, passed through the wound into the lower segment of the esophagus, and thus he was fed with the aid of a funnel and without regurgitation (Fig. 5). Another tube, its lower end tied off with a string, was passed into the upper segment of the esophagus for about five inches and left there to maintain patency. Five days after resection the wound was nicely granulating and a full-sized stomach tube passed through the mouth appeared in the wound.

About four days later a distinct, though slight, contraction was noted at the opening in the upper segment of the esophagus. An Einhorn tube was placed in the stomach through the mouth and through this the patient was fed for several weeks—milk, egg, and sugar being injected with a syringe.

January 12, 1921, I cocainized the pedicle of the skin-flap and divided it at the angle where the flap dipped into the chest. It was then dissected away from the inner part of the chest wall and turned over the esophagus so as to form a rude tube. (Fig. 6). It was hoped that the entire wound would now fill with granulations and that the case would soon be ended. The flap, however, proved to be most obstinate in its tendency to return to its normal position. During the next few weeks it had firmly adhered to the chest wall three times, in spite of every effort to hold it in place with sutures and packing, until finally I was forced to let it have its way for the time being.

On January 26, the granulations looked a little flabby, and antisyphilitic mixed treatment was begun. When the patient swallowed a glass of water about four drams of the six ounces entered the stomach.

February 8, the mixed treatment had been followed by an improvement in the appearance of the wound. For the next few weeks the patient was fed entirely by mouth, the Einhorn tube having been removed and replaced by a large tube, No. 44

caliber, which was passed through the wound into the openings in the esophagus so as to bridge the gap, and was kept from slipping into the stomach by a stout silk ligature which emerged at the margin of the wound, and was strapped to the patient's back. Through this tube he was able to take fluids and semi-

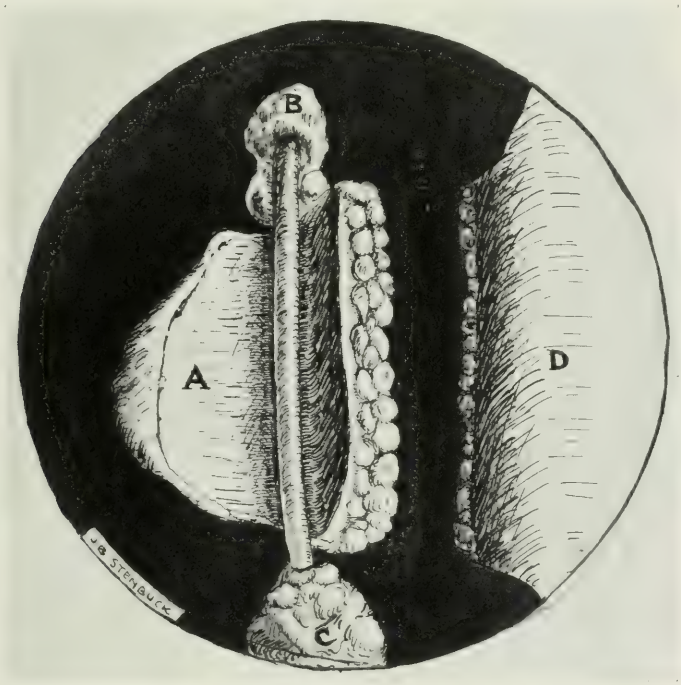

FIG. 6.—Skin flap cut through and rolled over a rubber tube toward the tip of the flap *A*. *D* is the pedicle of the flap, now no longer a part of it. *B* and *C* are the upper and lower esophageal openings bridged by the tube.

solids by mouth, and I hoped that when granulation should have been closed the defect the tube might be removed by way of the mouth through the esophagoscope. Some days later, however, this tube became disarranged and the patient strangled and was distressed so that it had to be removed through the wound. The man was rapidly gaining in weight and his general

appearance became normal. His wound was packed with rubber dam several times a day, which stopped the opening in the back, so that he now took all his nourishment by mouth, liquids and semisolids, with but little leakage, and I thought we were on the last lap of this trying surgical contest. It soon became evident, however, that obstruction at the end of the upper esophageal segment was increasing and it was more and more difficult to

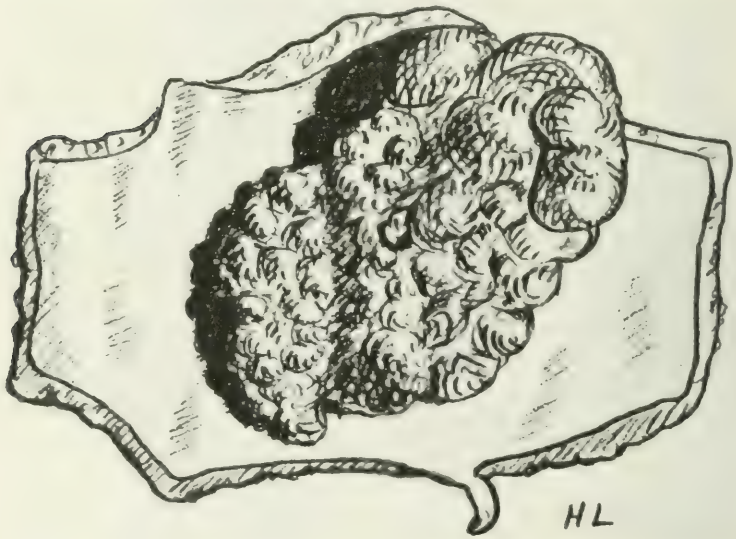

FIG. 7.—The tumor and esophageal segment laid open.

pass bougies. Evidently, too, there was a tortuosity in the new esophagus, and this was later demonstrated by the *x*-ray (Fig. 8.) Dr. Yankauer dilated the upper opening with the aid of the esophagoscope, but the straight instrument could not be made to pass into the stomach. Meantime the external wound was allowed to close until only a small fistula, say about No. 12 French, persisted, the remainder having cicatrized firmly; but because of the increasing difficulty in passing bougies from the mouth to the

stomach I felt that it would be necessary to reopen the wound, so, on March 31, without anesthesia, this was done. The new esophageal lining was exposed by cutting through the posterior

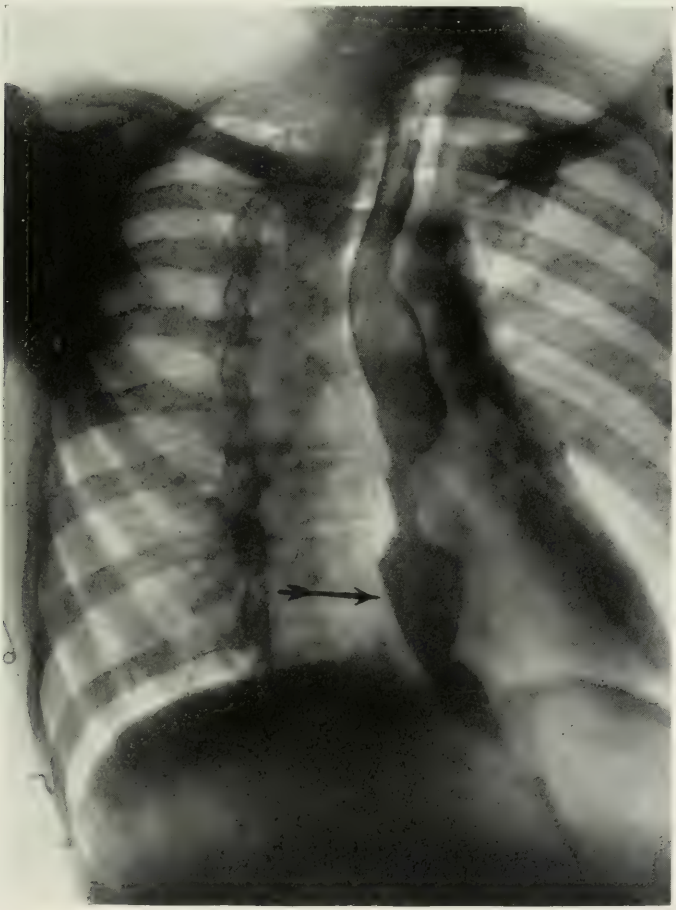

FIG. 8.—*X*-ray about three months after the operation. Thick barium mixture. The arrow points to the new esophagus (the part made from skin flap). This part is partially empty. Note stricture above it.
(*X*-rays from the Radiographic Department, Mt. Sinai Hospital, Dr. Leopold Jaches, director.)

line of union, which proved to be tough and cicatricial. It was with great reluctance that I sacrificed the skin union so long and anxiously awaited.

The cutaneous lining of the esophagus was white and looked normal, although in the neighborhood of the external wound there

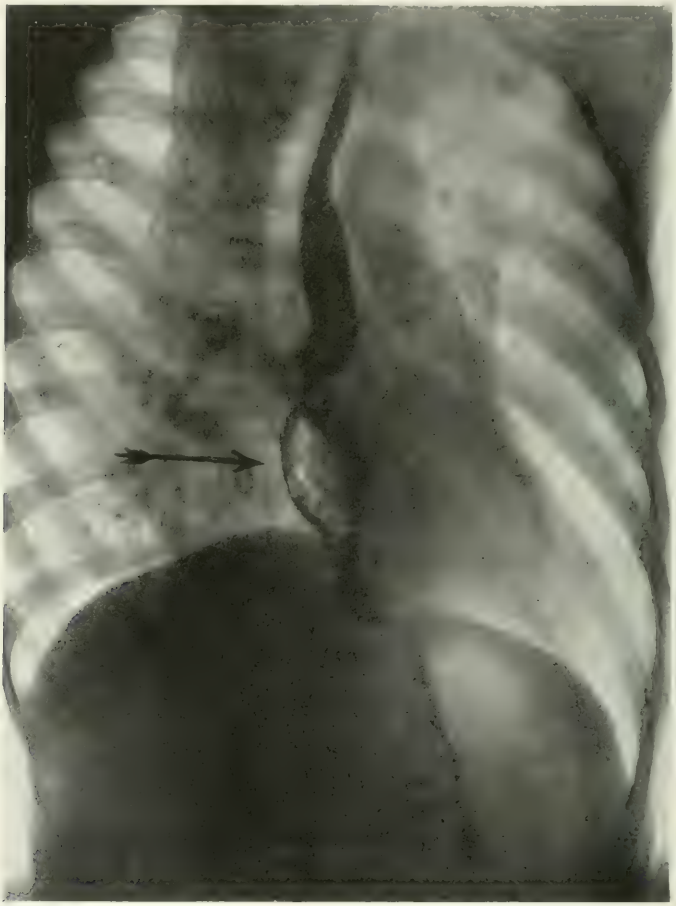

FIG. 9.—Five months after operation the wound is healed. Thick barium *x*-ray in oblique position as in Fig. 8. The stricture has been dilated. Arrow shows new esophagus.

was dermatitis and excoriation, evidently from the presence of decomposing food. The finger in the wound detected the upper and lower esophageal apertures, the lower easily admitting the

index finger, the upper being much contracted. It is possible that this phenomenon might be accounted for by disturbance

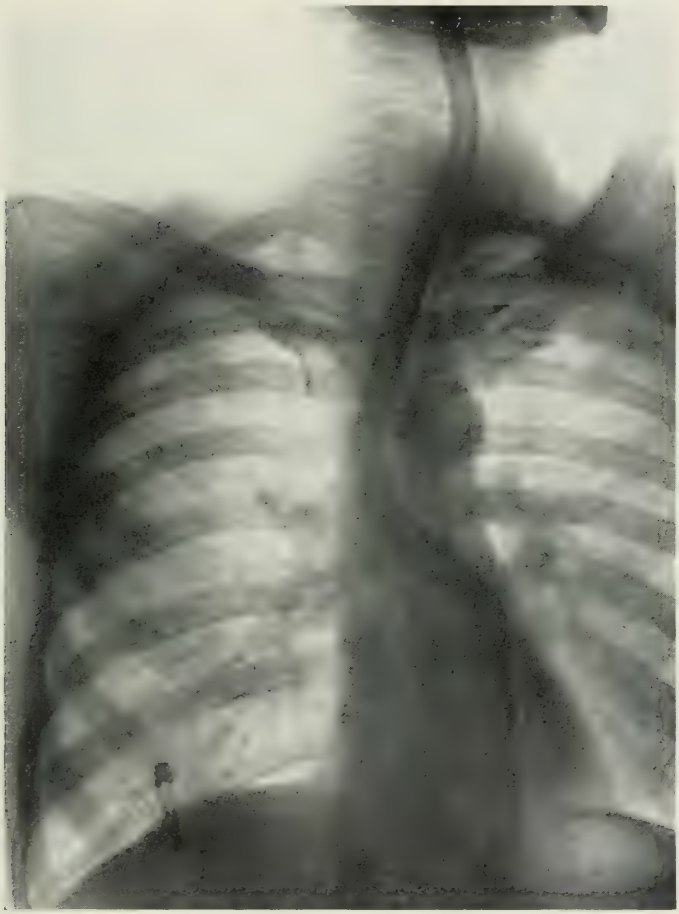

FIG. 10.—A red rubber stomach tube has been passed by the patient. In this oblique position the canal appears fairly straight.

of the nerve supply to the lower segment. A silk string was swallowed by the patient and a tightly fitting rubber tube was drawn through the upper segment of the esophagus and left

there to stretch the stricture for twenty-four hours, a small stomach tube being inserted through the cardia for feeding.

To go into all the details would be even more boring than this long paper has already become; so I will merely say that although

FIG. 11.—Anteroposterior view at same sitting as Fig. 10. Note bend in esophagus.

there was good dilatation by the tube, contraction again occurred in spite of the daily passage of instruments. The condition was enough to try one's patience to the utmost and I began to hear hints that a gastrostomy might simplify things. This it certainly

would have done, for it would have permitted the easy dilatation of the strictures with bougies tied to a swallowed string emerging at the gastrostomy wound. But I had set my heart on finishing the case without opening the stomach. Finally, I was persuaded

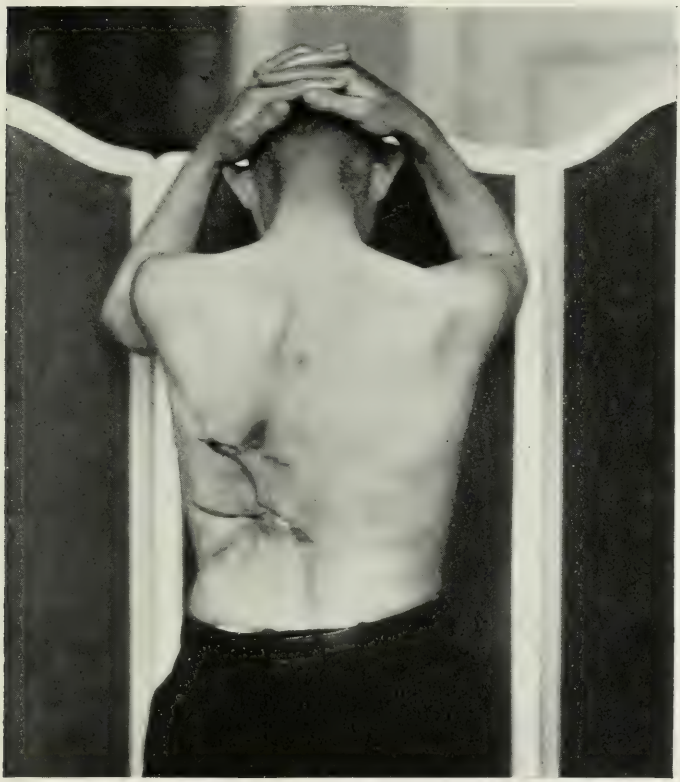

FIG. 12.—Final result, showing wound healed after plastic operation.

by Dr. Neuhof to reopen the wound and to expose the upper and lower orifices to inspection, dealing with them as might appear necessary. Accordingly I did this on April 28, without general anesthesia, but using a little cocaine locally. With retraction

there was perfect exposure of the two apertures. Both had united nicely with the skin, but the upper was a mere slit with a tough cicatricial ring forming a collar about it. With a probe-pointed bistoury several radiating incisions were made until the largest-sized bougie, about No. 50, could be easily passed without trouble. The lower opening was treated in the same manner and for the same reason, although the contraction here had been slight. A piece of the contracted skin was removed for microscopical examination. It showed nothing remarkable.

The patient then began to pass a No. 45 stomach tube himself, and I felt the time had come to close the thoracic wound. On May 5, I performed, in general anesthesia, what I hope will be the final operation in this case. Thoroughly denuding the edges of the wound in the esophagus, I closed it with interrupted chromic gut sutures, leaving a small opening below. I then dissected up the skin of the back, forming two flaps, one on each side of the wound, and sutured them together.

On May 11, healing had so far proceeded that the patient swallowed liquids naturally and without losing a drop. Four times a day he passed a No. 45 stomach tube. He could take ordinary food made into a mash or after thorough mastication. The wound had healed soundly in the depths and there remained but two small flat granulating areas on the back where the flaps had been slid at the last operation. He was discharged otherwise apparently well on May 19. It is intended that he shall pass the bougie several times a day for the greater part of a year or until there is reason to believe that the cicatrix will no longer contract. Davis F. was an ideal individual for pioneer work. If he was nervous, he never showed it, and he coöperated in every possible way, enduring pain and great discomfort without complaint. Also, he evinced, in a detached way, keen interest in the outcome of his case.

The history of this case is that of my first completed operation of extrapleural resection of the esophagus without gastrostomy, and I believe it is the first in medical history. Four other patients upon whom I have made exploratory mediastinotomies for car-

cinoma of the esophagus died, and in every case the tumor had extended beyond the esophagus. Three of the deaths were caused by infection of the mediastinum through injury to the diseased esophagus while attempting to isolate it. In the fourth, that of Mrs. U., I have not been able to explain satisfactorily the operative death, although reference to the postmortem notes will show that the case was inoperable. Abstracts of the four histories follow:

CASE I.—R. A., male, aged fifty-six years, entered Mt. Sinai Hospital, October 14, 1919. "For a number of months there had been vomiting and regurgitation of solid food immediately after swallowing, until finally only water could be taken by mouth." The patient was cachectic and emaciated, with rales at both bases. The stomach tube was obstructed 45 cm. from the teeth. *X*-ray examination showed complete obstruction at the lower and middle thirds of the esophagus.

October 30, 1919, a Witzel's gastrostomy was performed by Dr. Neuhof. Specimen removed by Dr. Yankauer through esophagoscope reported by Dr. Mandlebaum "squamous cell carcinoma." Mediastinotomy was done on November 13, 1919, by me. Intratracheal anesthesia by Dr. Branower. The long posterior eighth interspace incision was made, and the eighth, ninth and tenth ribs were divided behind their angles. Owing probably to previous pleurisies there was complete obliteration of the left pleural sac by adhesions. This facilitated the operation. The flap of chest wall made by the incision just described was turned down and the aorta easily exposed for at least eight inches. The esophagus was located with a bougie passed through the mouth and the tumor was easily reached. It was much larger than expected and was adherent to the pericardium and lower part of the left lung. The lower limit could not be ascertained because the growth seemed to pass through the diaphragmatic opening. The adherent lung was divided between ligatures and the esophagus above the tumor was mobilized after cocainization of the vagus. Pus was encountered close to the tumor and the

operation abandoned. The wound was closed with drainage posteriorly. No shock followed the procedure. Two days later the wound was dressed, the patient sitting up in bed without assistance. There was infection, however, from the suppurating tumor and death occurred four or five days after the operation. No autopsy.

Case II.—John F., aged fifty-six years, had had great difficulty in swallowing solids for only six weeks. When he was admitted to Mt. Sinai Hospital there was extreme emaciation and the *x*-ray showed esophageal obstruction opposite the seventh dorsal vertebra, with a filling defect extending downward for three inches and with dilatation above. There was inequality of the pupils, but the Wassermann test was negative. On June 24, 1920, Dr. Neuhof assisting and Dr. Branower administering the anesthetic by the intrapharyngeal method with ether, I operated. A long seventh interspace incision curved downward posteriorly over the eighth, ninth and tenth ribs. The eighth rib was resected subperiosteally and the ninth and tenth ribs divided posteriorly through the periosteum, the finger holding the pleura off from the ribs. The pleura was then stripped further away, exposing the posterior mediastinum, but it was wounded in several places and not sutured. The exposure was excellent with the aid of the rib-spreader and the neoplasm was easily found. There was an infected gland, and considerable time was spent in dissection. The tumor was firmly adherent to the aorta and the operation was discontinued. In dissecting the esophagus with the stomach tube in place the musculature of the wall was injured so that the tube was clearly seen through the mucosa, and eventually it was shown that infection took place here. The wound in the chest wall was closed by suture, the skin being left open. Two days later there was but little shock and the patient swallowed fluids better than before, owing to the stretching of the esophagus by the stomach tube. After that, however, general wound infection rapidly progressed and he died a few days later.

Case III.—Samuel R., aged sixty-eight years, entered Mt. Sinai Hospital March 2, 1921. He had had "dropsy" thirteen years before. For two and a half months there had been regurgitation of food, with difficulty in swallowing and pain in the epigastrium and lower sternal region. The patient was obviously an old and emphysematous man. The *x*-rays showed almost complete obstruction of the esophagus near the stomach. March 3, 1921, gastrostomy was performed by Dr. Ira Cohen, adjunct surgeon. Pneumonia followed, although local anesthesia had been employed. Then there was a double suppurative parotitis, which finally yielded to massage. At last, on April 4, 1921, it was considered safe to perform the mediastinal operation. Nitrous oxide and ether anesthesia.

Because of the complete obstruction in this case, continuous suction was carried out with a small-sized esophageal tube, which was inserted as far as the stricture. To make the suction more effective a dozen perforations were punched out of the wall of this tube. The object of this suction was to get rid of the fluids which might have entered the esophagus, gradually filling it up to a point of overflow and causing aspiration pneumonia. The long flap was isolated and the first steps of the operation were similar to those described in the successful case. The pleural cavity was unavoidably entered in several places, the operation proceeding, however, under intrapharyngeal insufflation. Two ribs above the ninth, and also the tenth rib, were divided posteriorly, the pleura being here easily stripped away. Working upward, now, I was surprised to see the entire posterior pleura as far as the midaxillary line fall away from the costal and mediastinal chest wall. The pleura, however, was as fragile as wet tissue-paper and was torn many times. The aorta and great splanchnic nerve were seen, the esophagus palpated, and the tumor easily found. The upper limit was sharply marked about two inches above the diaphragm, but the lower limit was not reached, although it was believed to be above the diaphragm. While attempting to isolate the normal part of the esophagus the right pleura was suddenly entered. There was a sound of rushing

air, but the opening could not be seen. The heart's action became very irregular, the patient turned cyanotic, respirations were shallow, and at last it looked as though the man were dead. Thinking that the cardiac collapse was perhaps due to vagus disturbance a pledget of cotton with 5 per cent cocaine solution was placed for a few moments in the wound over the nerve and the wound in the chest fully closed. To my surprise the patient gradually recovered so that he left the table in good condition. About forty hours after the operation, however, he sank and died.

The postmortem inspection showed that the tumor had invaded the stomach, although that had not been ascertained at the time of gastrostomy. The right pleura had been entered and contained air and bloody serum. Death was apparently due to cardiac disturbance caused by pneumatic and circulatory changes in both chests. Pathological report of tumor by Dr. Mandlebaum was "adenocarcinoma with pearl formation."

Case IV.—Mrs. L. U., aged forty-four years, had suffered from palpitation and dyspnea on exertion for several years. She had also had an attack of edema of the extremities seven months before I saw her. She entered Mt. Sinai Hospital on April 12, 1921, with a temperature of 101° F., pulse 84 and respirations 24. For six months there had been dysphagia and vomiting and loss of weight. There was much pain in the chest on deglutition. The patient was badly nourished and poorly developed. There were rales at the left apex with slight dulness. There appeared to be some cardiac enlargement with a systolic murmur at the apex. The spleen was palpable. The urine showed nothing abnormal. By the *x*-ray there was great narrowing of the esophagus at the level of the seventh vertebra, with a defect below. The patient could take the thick barium mass, but it passed through the stricture slowly. On April 13, by esophagoscopy, Dr. Kaempfer removed a tiny specimen which twenty-four hours later was reported by Dr. Mandlebaum as probably carcinoma, though the specimen was hardly sufficient for complete examination. Bleeding had greatly embarrassed

the esophagoscopy, but Dr. Kaempfer said that inspection strongly suggested carcinoma.

On April 14, operation was performed. Dr. Branower administered nitrous oxide and ether by the intrapharyngeal method and Dr. Neuhof assisted at the wound. A perforated stomach tube with suction was used to keep the esophagus free from fluid. The skin flap was made as in the first case with its midline along the seventh rib, and about five inches of this rib were then resected subperiosteally and the pleura peeled away without injuring it. The sixth, fifth and fourth ribs were divided near the spine, and later the eighth rib as well. The great splanchnic nerve and the aorta were plainly visible. The tumor was found. Its upper limit was about one and a half inches below the aortic arch, but its lower limit was not palpable. No instrument could be passed through the stricture to facilitate the identification of the lower esophagus. The upper part of the growth was firmly adherent to the aorta and it was peeled away with difficulty, this procedure being preceded by cocainization of the vagus. It looked as if small portions of the tumor remained adherent to the aorta. Further dissection showed that the growth could not be freed from the pleura, so, fearing that I might wound the already eroded esophagus, the operation was abandoned, though I hoped that it might be completed at some future time. The patient died, however, thirty-three hours after the operation after a period of deep cyanosis with loud rattling in the trachea.

The postmortem examination through the wound showed a large ulcerating carcinoma which had completely and widely perforated the esophagus and was on the point of entering the pleura; indeed, there were pleuritic adhesions here with a few flakes of fibrin and a minute quantity of turbid exudate.

Conclusions. From the experiences outlined in this paper, together with six other cases of transpleural exposure of the esophagus with one recovery, a patient with esophagospasm which was later dilated, (*Ann. Surg.* 1916, vol. 64, p. 94,) I have reached the following conclusions:

1. That transpleural resection of the esophagus has a forbidding mortality.

2. That fatal infection follows the primary opening of the esophagus within the mediastinum.

3. That it is feasible to make an extrapleural exposure of the posterior mediastinum large enough to permit the operator to see clearly and to work safely with both hands.

4. That resection of the esophagus in the posterior mediastinum can be done by dividing the operation iuto two stages; at the first the esophagus is freed from its attachments and the mediastinum is sealed; at the second, ten to fourteen days later, the resection is performed.

5. This procedure deserves a fair trial by thoracic surgeons.

Operative Procedures and Suggestions from the Literature

Dorsal Mediastinotomy

Nasiloff (*Vrach.* 1888, No. 25) resected the third to sixth ribs on cadavers to expose the mediastinal portion of the esophagus extrapleurally.

Quenu and Hartmann, working on cadavers, did subperiosteal resection of portions of the third, fourth and fifth ribs.

Potarca discusses which is the better side for approach.

Bryant builds a quadrilateral flap. The middle rib of the flap is entirely resected, the other ribs temporarily resected. (Experimental only.)

Obalinski discusses the side of approach.

L. Rehn builds a large skin and muscle flap from the spinous process of third to ninth dorsal vertebræ; resects several ribs.

Von Hacker discusses side of approach, as does also Bourienne.

Note.—The first experiments on animals were made by Nasiloff. After this a number of surgeons operated not only upon dogs and human cadavers but also upon the *living human subject*. Rehn, Llobett, Tuffier, Faure, Küttner also tried

these methods, but none of their patients survived; all the operations were extrapleural.

Esophagoplasties. (*Not Resections.*)

Wullstein, 1904, mobilized a piece of jejunum far enough under the skin of the thorax to complete an esophagojejunostomy with the upper end of the esophagus. An external plastic; no resection of the esophagus. (*Deutsch. med. Wchnschr.*, 1904, No. 20.)

Roux, 1907, esophagojejunogastrostomy. (*Semaine med.*, 1907, iv.)

Herzen, employed a modified Roux method and reported a success. He shortened the path of the jejunum by a slit through the mesocolon and gastrocolic omentum and brought the gut up through this to the subcutaneous chest. (Anterior.)

Bircher, 1894 and 1907, connected the esophagus in the neck with the stomach through a tube made of cutaneous tissue—an esophagodermatogastrostomy. A fistula formed, as was also observed by Payr in a similar operation. (*Centralbl. f. Chir.*, 1907, No. 51.)

Esser, 1917, tunnelled a path in the subcutaneous tissue and used Thiersch grafts; the grafts were put around a glass tube. No note of end results.

Kelling, 1914, used the transverse colon; esophagocologastrostomy. Failure. (*Centralbl. f. Chir.*, 1913.)

Lexer, 1911, combined various types. Esophagodermatojejunogastrostomy or esophagodermatocologastrostomy.

Hirsch, 1911, used a pedicled flap made from the entire thickness of the anterior wall of the stomach.

Jianu, 1912, made a flap of the greater curvature of the stomach containing the left gastroepiploic artery.

Rehn and Röpke proposed making use of the stomach plus the healthy distal stump of the esophagus, after resecting it in the middle. Modification of Jianu.

v. Fink, first, then Enderlen and Hotz mobilized the stomach, closed the distal portion of the duodenum, did a gastroenter-

ostomy; then brought the duodenal end of the stomach subcutaneously into the chest wall.

Kirschner mobilized the entire stomach and brought it up into the anterior chest wall subcutaneously, making a gastrostomy opening in the chest, then connecting the stomach with the cervical portion of the esophagus. (Kirschner's article reviews to 1920 the work in esophagoplasty.)

Hofer and Kofler, 1917, built a skin flap with its base at the vertebral column, and later made a tube of esophagus. This work was done on the cadaver only.

Budde, 1921, built a tube of skin fascia and muscle around a glass rod; then implanted the tube between the cut ends of the esophagus extrapleurally. This was done on the cadaver.

Faure, Llobet and Tuffier all attempted to resect the esophagus extrapleurally, but failed.

Other Procedures

Jianu-Röpke, Gastrostomie und Oesophagoplastik. A tube was fashioned from the greater curvature of the stomach but left attached at the fundus, this tube being brought up under the skin almost as high as the clavicle; an end-to-end anastomosis with the upper end of the esophagus. *Deutsch. Ztschr. f. Chir.*, 1912, cxviii, s. 383.

Kirschner, four cases.—Two for stricture: one, an esophagogastroplasty for stricture, alive and well; the second, a stenosis of the esophagus with lung abscess, died of lung complications and infection fourteen days after operation. There were two cases of carcinoma of esophagus: the first, incision in the seventh left intercostal space, intrapleural, inoperable because of metastases; the second died of mediastinitis.

Halsted. Tracheotomy; insufflation of lung; incision in seventh interspace; rib-spreader used. (*Jour. Exp. Med.*, 1909, 373–391. Work on thoracic aorta.)

Enderlen made a rectangular flap with its base to the spinal column from the spinous process of the third to the ninth dorsal

vertebra and extending to the mesial aspect of the scapula. All soft parts were divided down to the ribs and turned back. The periosteum was dissected away from the ribs, and pieces of rib, 10 cm. in length, were resected.

Henle and Enderlen, operated for removal of foreign bodies—dorsal mediastinotomy. Henle's patient died nine days postoperative. Enderlen's patient was discharged cured after nine months.

NOTE.—No surgeon operating for noncancerous stricture of the esophagus attempted resection. All made use of external esophagogastrostomy or some other complicated procedure.

SOME CONTRIBUTIONS TO THE SURGERY OF THE ESOPHAGUS AND POSTERIOR MEDIASTINUM

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Ach: Beiträge zur Oesophaguschirurgie, Habilitationschrift, 1913.

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DIAPHRAGMATIC HERNIA; THE THORACIC APPROACH

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THE essential purpose of this communication is to call attention to a type of diaphragmatic hernia occurring in the pedestrian child by the passage of an automobile wheel over the upper abdomen, and to consider the methods of cure by operation.

A review of the literature on hernia of the diaphragm shows a substantial and progressive increase in the number of cases discovered and cured. It is evident that internists and roentgenologists in particular are on the alert for it. The unexpected is less frequently met at operation and there is abundant evidence to indicate that the mortality figures have been reduced strikingly within a decade.

Scudder[1] found 53 operated cases recorded up to 1913. Frank[2] carefully reviewed the literature in 1919 and could add only 41 additional cases over a period of about seven years. From 1918 to 1920 inclusive, 96 cases have been reported. It is probable that in my search of the literature there has been some overlapping of the series recorded by Frank. Nevertheless, the rapid increase in the number of operations for this deformity is very manifest. Of the 96 cases collected 43 were the results of battle casualties. There can be no doubt that the number of cases treated and not reported is considerable. The number of cases observed among children is singularly small, 5 in all; 3 were of congenital origin and 2 reported in this article were caused by external violence.

In diaphragmatic hernia intestinal obstruction is the outstanding factor of risk to life. In Scudder's series the surgical mortality was 75 per cent, essentially because of intervention during strangulation of the transposed viscera. Unlike this accident in other types of hernia the process is concealed and the symptoms and physical signs somewhat strange. Hence, delay is the rule and the mortality exceedingly high. The operative mortality in any series depends upon the number of cases encountered during acute intestinal obstruction. Warren[3] reported 8 cases treated surgically during ten years at the London Hospital. Only 1 lived. 7 were operated upon in the presence of acute obstruction.

From current reports and observation it appears that most patients, even children, withstand surgical intervention for repair of the diaphragm remarkably well. The general mortality after operation in the non-strangulated type of diaphragmatic hernia averages below 10 per cent. It should be less than 5 per cent when experience is enlarged and the methods of intervention best suited to each case are more clearly defined.

Innervation of the Diaphragm. The primitive mammalian diaphragm is derived from the muscular myotomes which invade the septum transversum. It becomes a muscular partition with an important innervation. In its descent from its position under the fourth and fifth cervical segments the muscle buds carry their nerve, the phrenic, with them. The entire nerve supply of this musculofibrous septum has been a subject of absorbing interest to anatomists and physiologists for more than a century. More recently the attention of surgeons has become centered upon a consideration of this phase of the subject because of the congenital defects and injuries of the diaphragm which require operative measures for repair.

The left phrenic nerve, that in which we are particularly interested because hernia is at least eight times more common on the left side, pursues a general anterolateral course and pierces the diaphragm at the junction between the musculature and the central tendon. Under cover of the peritoneum, it splits into an anterior, a lateral and a posterior branch. The anterior

supplies the muscle of the left sternal portion and the antero-lateral part of the left costal portion. The lateral branch supplies the corresponding part of the left costal section. The posterior branch is distributed to the left lumbar region of the muscle.

In its descent to its permanent level, according to Keith,[4] the diaphragm receives muscle tissue from the transversalis and the rectus sheaths, taking with it an innervation from the thoracic segment of the spinal cord. This innervation of the human diaphragm from the lower intercostal nerves was discovered almost a century ago by several of those accurate observers, the dissecting anatomists. Meckel[5] described it in 1817. Joseph Swan[6] gave a very explicit account of this innervation in 1830. He described filaments to the diaphragm from the sixth, eighth, ninth, eleventh and twelfth intercostal nerves.

The careful histological studies of Pansini,[7] who was the first observer to reinforce the dissection method by histological examination, and of Cavalie,[8] from a dissection of twenty human diaphragms, confirmed the work of the early observers and proved beyond doubt the existence of an intercostal innervation to the diaphragm. Keith[9] has shown that in an African toad, the Xenopus, the two nerves which represent the phrenic nerves of mammals come off from the second and third spinal nerves; these two nerve branches pass respectively to the ventral part of the amphibian diaphragm and to its dorsal portion. In all birds, except the ostrich, the innervation of the diaphragm is from the intercostals. The ostrich is the only bird which has retained part of its phrenic innervation. Thus, in respect of the nerve supply of the diaphragm, mammals have retained the cervical innervation of their amphibian ancestors. Except its rim, which is supplied by filaments from the seven lower intercostal nerves, Kidd[10] believes that the entire diaphragm derives its pain-conducting nerves from the phrenics. He is of the opinion also that the thoracic innervation is both sensory and motor and comes into action during deep and forced inspiration.

Operations for hernia of the diaphragm have been carried out with little consideration of the permanent, partial functional

derangement of the affected half of this septum from rupture, especially that form which results in lacerations of considerable degree. In rare cases where the nature and extent of the defect or laceration is such as to involve one or more of the large branches of the phrenic nerve, it is inevitable that the functional capacity of the affected side of the diaphragm rarely, if ever, returns to normal. In the case to be recorded the diaphragm is shown to have a wider range of mobility than normal, months after operation. This would indicate defective innervation and a high diaphragm comparable to a moderate degree of eventration.

That part of the diaphragm which is essentially muscular forms its rim. The central portion is fibrous. Under these circumstances, and from a surgical viewpoint, the complete innervation of the diaphragm becomes an important consideration. Admitting that the main nerve supply is from the phrenic, the preservation of an associated innervation such as it derives from the intercostal nerves is a factor to be considered in the transthoracic operation.

Methods of Operation. Both the clinical and structural picture of diaphragmatic hernia vary widely and the method of operation best suited for its repair necessarily remains a problem to be decided upon the evidence in each case.

There are two methods for approaching the diaphragm, the abdominal and the thoracic. Most of the operations reported by American and British surgeons show a decided preference for the abdominal approach, while the French favor the thoracic operation. The thoracic cavity is entered so seldom in comparison with the abdomen that, when a choice of approach is to be made, it naturally requires strong inducements to divert the average surgeon from the transperitoneal route. Then again, so dissimilar are the cases of diaphragmatic hernia in certain particulars that no one plan of approach is best suited to overcome all barriers. Consequently another method which combines the thoracic and abdominal approach has been practiced by Auvray[11] and other French surgeons. It consists of a resection of the ninth rib from behind forward to its anterior end, and the incision car-

ried down to the umbilicus. The cartilaginous margin is cut through and the diaphragm incised to the hernial opening. The entire opening in the diaphragm is then sewed up and the wound closed. Auvray terms this procedure a thoraco-laparo-chondro-phrenotomy.

Invasion of one side of the thorax in liberal fashion has become a more common operation since the war, and it is fair to assume that the tendency toward hesitation at the thought of thoracotomy will disappear as operations on the thorax increase.

In a monogram on surgery of the chest from battle wounds, Sir Berkeley Moynihan[12] makes the following comment: "The subject of thoracic surgery is one which before the war had been made difficult by the cumbersome methods employed. The fear of pneumothorax was present in the minds of most surgeons and was a powerful deterrent." However, Bazy, of Paris, had spoken of the safety of open operations on the chest for years before the war. Cranwell[13] was among the first to suture the diaphragm successfully by a transthoracic approach. We have evidence that American surgeons were of the same opinion. Binnie,[14] in the second edition of his volume on surgery, advocated the transthoracic operation in 1906. Carson[15] adopted this method of approach in 1912. Two cases of the delayed incarcerated type were operated upon by Stuart McGuire, of Richmond, Va., in 1913.[16] In a personal communication from Dr. McGuire I have learned of a third case which he has cured by the transthoracic operation.

In order to accomplish the desired end, Frank[17] found it necessary to abandon the attempt by laparotomy and at a second operation successfully closed the aperture in the diaphragm through an opening in the chest wall. Greig, of Edinburgh,[18] has recorded an unsuccessful attempt by the abdominal approach to withdraw the herniated stomach from the thorax on account of adhesions. Downes operated upon a child of seven years by abdominal section. In addition to pyloric stenosis he found the congenital type of hernia. He met with such difficulties that he deemed it unwise to attempt reduction and suture of the dia-

phragm. Gastro-enterostomy was done and a good functional result accomplished. Major C. D. Granger, of the British Army,[19] met with extreme difficulty in suturing the aperture in the dia-

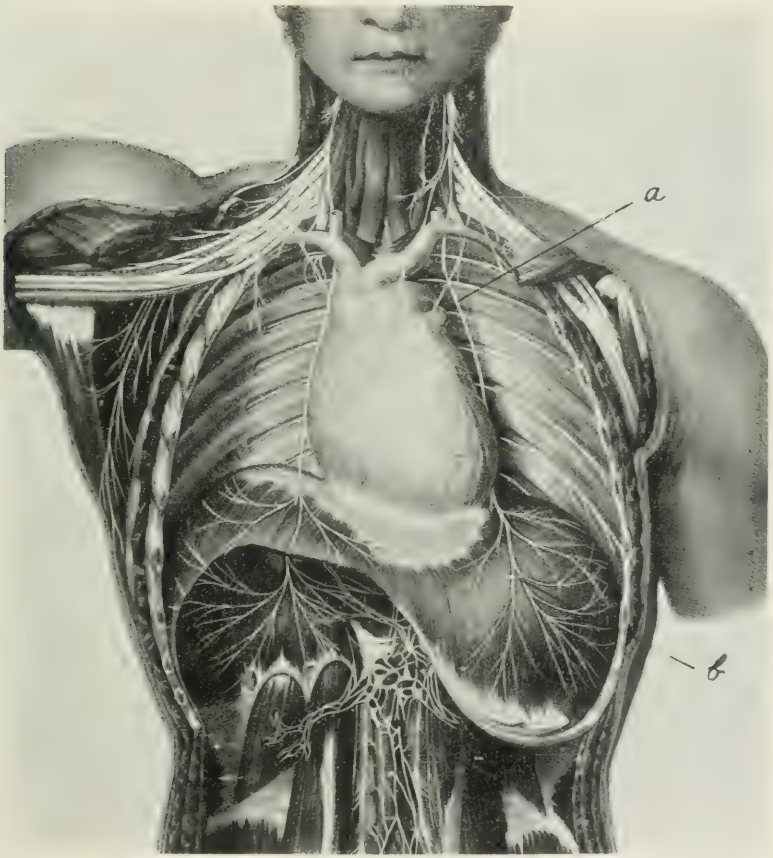

FIG. 1.—Showing (*a*) the course of the phrenic nerve, and (*b*) its distribution where great damage may result to the innervation of the diaphragm from rupture.

phragm through an abdominal incision. He was unable to complete the operation satisfactorily after two hours' work on account of the collapsed condition of the patient. A case has been reported

by W. J. S. Bythell[20] in which an unsuccessful attempt was made by Mr. Howson Ray to deal with the herniated stomach through an abdominal incision in a child of nine years. At the first operation he merely sutured the stomach to the abdominal wall.

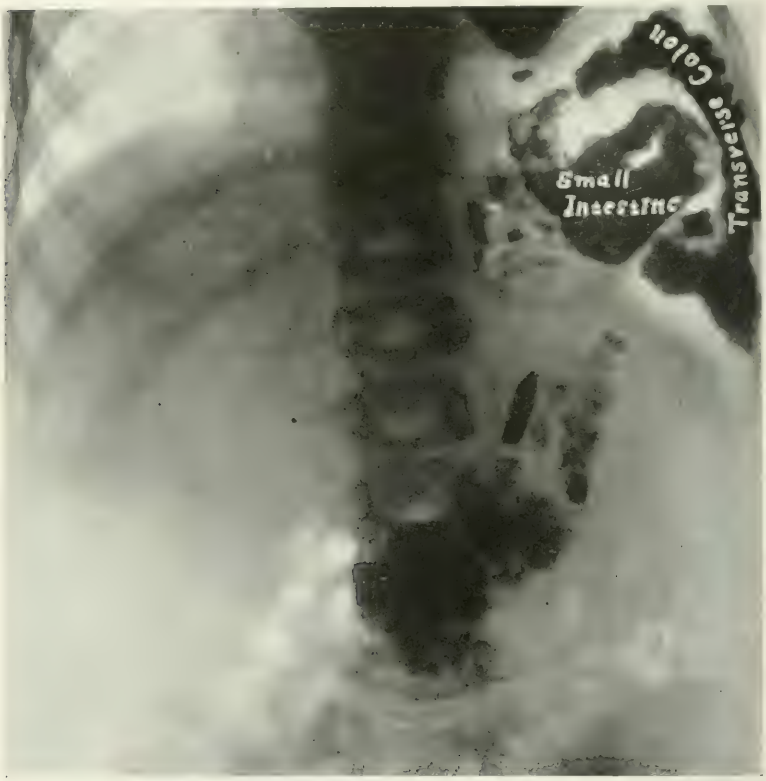

FIG. 2.—Roentgenogram showing herniated abdominal viscera in the left pleural cavity.

Apparently an attempt to close the gap in the diaphragm appeared to be inadvisable. The hernia soon recurred, and after a few weeks a gastrostomy was performed. Andrews[21] describes an operation during which he found dense adhesions of the omentum within the thorax. He was unable to separate these

from below without considerable risk, so he sectioned the omentum and left it in the pleural cavity. Wiart,[22] in working from below, found it impossible to close the opening in the diaphragm completely. Weiss and Dujarier[23] found the abdominal approach so difficult that they were at the point of abandoning the operation.

A case reported by Jopson,[24] seen in consultation at Base Hospital, No. 18, A. E. F., was dealt with successfully by thoracotomy after a second attempt by laparotomy had failed to relieve attacks of intestinal obstruction.

Thus in scanning the literature one finds ample evidence to show that in a good proportion of cases the abdominal approach is by no means ideal and probably is beset with more difficulties and dangers than the transthoracic way.

Among the advantages which may be claimed for the thoracic operation are the following: (1) Thoracotomy affords a more direct approach. (2) Pneumothorax and collapse of the lung are not to be feared. They exist already, as inevitable consequence of a hole in the diaphragm. (3) The open thorax allows the separation of adhesions under the eye of the operator. (4) Once the herniated viscera are replaced within the abdomen, they are out of the way, warm and well protected. (5) There is less shock from the thoracic operation. (6) If a hernia sac exists it can be disposed of in the most advantageous manner. (7) Operating on the convexity is more natural and more simple than suturing within the concavity of the diaphragmatic dome.

Based on these premises the transthoracic approach to the diaphragm appears to be the more logical and sound. However, a single experience or observation is sometimes all that is necessary to demonstrate how easily one may be led to miscalculate on the virtues of a chosen method. In order to make this evident by demonstration I shall record two cases of diaphragmatic hernia in young children. Each was produced in the same manner, *i. e.*, by the passage of an automobile wheel over the upper abdomen. The two cases were comparable in their important features with a single but remarkable exception. One was

operated upon by the transthoracic approach, the other by laparotomy.

Case I. L. S., a boy, aged five years, was run over by an automobile on May 11, 1920. He was taken to a hospital, where examination showed that he was severely injured internally, but the exact nature of the trauma could not be determined. In December, 1920, he began to have attacks of a peculiar nature resembling intestinal obstruction. These increased in frequency and severity. On January 4, 1921, after passing through the hands of many doctors, he was examined by Dr. Henry I. Bowditch and Dr. John T. Bottomley, of Boston, who made the diagnosis of diaphragmatic hernia. Later this was confirmed by roentgenologic examination by Dr. Ariel George.

The boy was admitted to our clinic for operation on February 23, 1921. With all the evidence required in our possession, it was obvious that surgical intervention was urgent. After due consideration of the abdominal and thoracic methods of approach, it was decided to attempt a reduction of the displaced organs and a repair of the opening in the diaphragm by the thoracic route.

Anesthesia. Although the child showed no evidence of organic disease, he was anemic and obviously asthenic. This lowered state of his endurance and the stress of the proposed operation gave accent to the importance of the choice of anesthetic and the manner of its administration.

The anesthetist, Miss A. M. Hunt, employed the method advocated by Sir Berkeley Moynihan, omitting his preliminary hypodermic injection of morphia, scopolamin and atropin. Nitrous oxide gas was employed for induction, shifting promptly to ether and oxygen. From the beginning the anesthesia was pushed to a considerable depth, allowing complete muscular relaxation. This was accomplished without cyanosis. From the inhalation of an excess of oxygen, a bright red color of the child was maintained. The closed inhaler of Connell was used, thus reducing the required amount of ether for the operation to about

an ounce. The range of respiration was kept shallow. This partial control of motion in the thorax greatly facilitated reduction and suture.

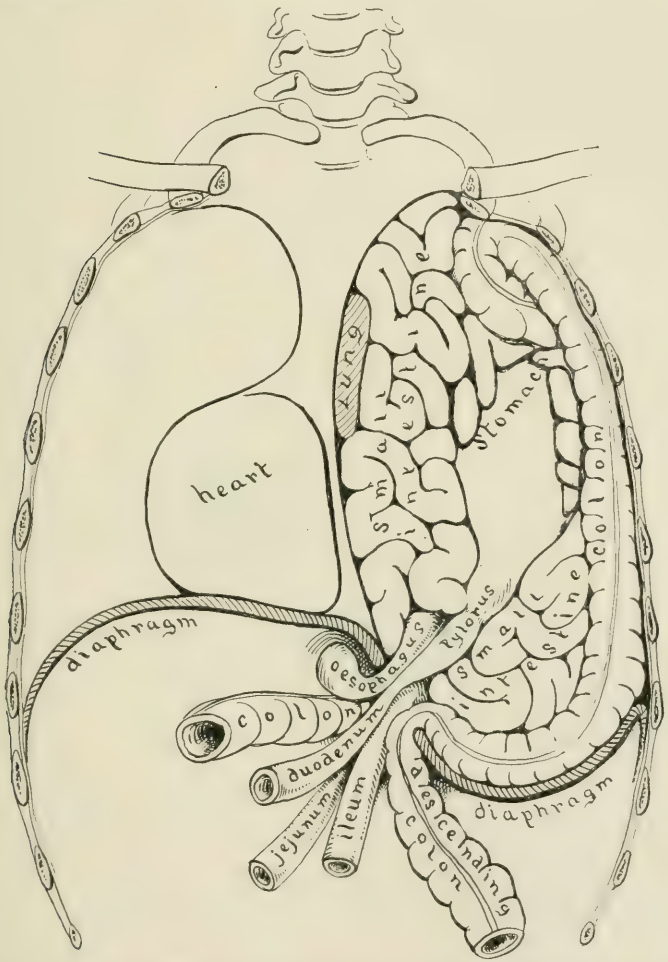

FIG. 3.—Diagram showing transposition of abdominal organs into left pleural cavity in Case I.

Operation. A lapel incision (Cranwell) was made through the soft tissues over the seventh and eighth ribs. The pleural cavity

was opened by an intercostal incision the entire length of the shaft of the rib. The seventh and eighth ribs were each severed in two places, first at the angle and then at the sternal extremity. Turning the seventh rib upward and the eighth downward gave a

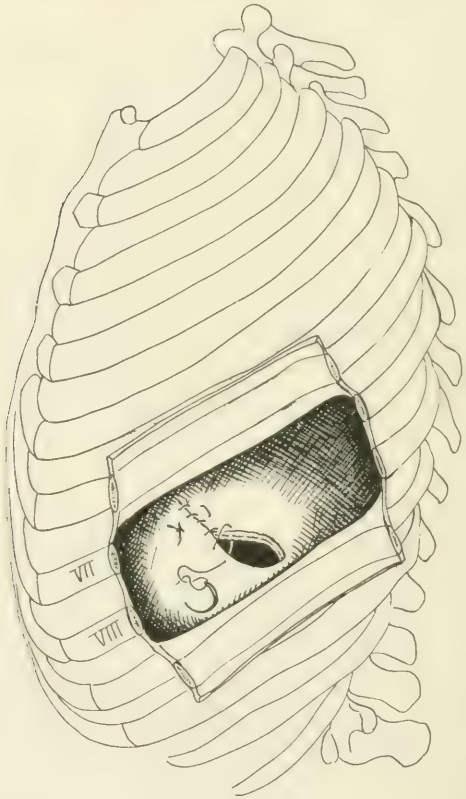

FIG. 4.—Trap-door opening in thoracic wall and suture of aperture in the diaphragm.

double trap-door opening into the left thoracic cage. As soon as the pleura was incised, distended loops of small bowel escaped and could be reduced only after the intrathoracic pressure was minimized by spreading the wound wide open with rib retractors. The chest cavity was filled with stomach, small and large bowel.

The left lung was collapsed against the spine and resembled the spleen in appearance. The diaphragm was prolapsed. Near its central attachments was an opening occupied by the herniated abdominal viscera. There was a close grip by the aperture upon its contents, and engorgement of the circulation indicated that strangulation was impending as a result of the stricture. There were no adhesions and no sac. Efforts to replace the intestine within the abdomen manually were encompassed with difficulties. As fast as the bowel was passed downward through the opening it was sucked up again. Repeated efforts resulted in failure and no progress whatever was made until the diaphragm was elevated to a much higher plane by placing two fingers under the outer margin of the hernial ring. Thus a partial vacuum was created within the abdominal cavity, and the small gut, the transverse colon, and finally the stomach were pushed through into the abdomen by the use of long, curved, blunt forceps. Once restored, it was not difficult to keep the abdominal viscera under the diaphragm by the use of a gauze pad. The remainder of the operation was comparatively simple. Exposure of the aperture in the diaphragm was ideal. The gap began a few centimeters posteriorly and external to the esophageal opening and extended in an oblique direction forward and outward. A moment's inspection showed that it did not involve any of the normal openings. It was somewhat elliptical, with the long diameter about four inches. The edges of the opening were rounded and thickened. Closure was accomplished by a continuous suture of No. 2 iodized catgut reinforced by several interrupted deep sutures of the same material. The thoracotomy wound was closed tight with catgut and several mass sutures of silkworm gut. The little patient withstood the operation remarkably well and made an uneventful recovery, leaving the hospital on the fourteenth day.

Case II. Was seen at the Rhode Island Hospital in the service of Dr. J. C. O'Connell, May 21, 1921. The patient was a boy, aged three and a half years. He was run over by an automobile April 28, 1921. Admitted to the Rhode Island Hospital

in a state of shock. The chest signs were very unusual. Hernia of the diaphragm was suspected by Dr. R. G. Bugbee. A barium enema was administered and a roentgenogram showed the colon in the left thoracic cavity. There was a small hernia of the abdominal wall under the left costal angle produced by the weight of the wheel in passing over the child's body. Dr. O'Connell decided to approach the diaphragm by laparotomy and made his incision over the site of the hernia. Upon investigation he found that the stomach, jejunum, transverse colon and left lobe of the liver had passed through a large opening in the middle of the diaphragm. Another laceration extended transversely across the anterior third, involving the right wing of the diaphragm and opening the pericardium for a distance of about 5 cm. The heart could be seen plainly and at times dipped through the opening into the abdominal cavity. Both lacerations were closed by interrupted catgut sutures. Dr. O'Connell reports that the boy has made a good recovery and the operation was a success, as shown in subsequent roentgenograms.

The surprise in this case was the laceration extending across the diaphragm to the right side and laying open the pericardium. It might not have been overlooked during an approach from above, but there is a fair—if not a big—liability that it would have passed unseen. This case accentuates the importance, when operating through the chest wall, of palpating the underside of the diaphragm as far as the hand can reach.

Aside from the advisability of using the abdominal approach whenever it is thought necessary to operate on a hollow viscus, it must be conceded that another strong argument, if not an incontestible one, in favor of laparotomy, is the opportunity thus afforded for examination of the entire diaphragm.

Operation for the cure or relief of diaphragmatic hernia during childhood is rarely found in literature. Many text-books on pediatrics fail to mention the subject. Holt describes a case of diaphragmatic hernia observed in a child sixteen months old who died of pneumonia at three and a half years. In concluding his notes the author states that the condition is not amenable

to treatment. The case recorded by Downes and that reported by Bythell have been noted already. They were of congenital origin.

That the traumatic cases should be operated upon as soon as conditions warrant there can be no question. One should be able to make the diagnosis promptly if the physical signs are carefully noted with diaphragmatic hernia taken into consideration. Nevertheless, immediate operation is inadvisable, because a state of shock exists as a result of lung collapse and pneumothorax, not hemorrhage. The amount of bleeding from the torn edges of the diaphragm is probably slight

A case reported by Balfour, although an adult, was of the nonstrangulated type of hernia of the diaphragm in which the diagnosis was made prior to operation. In many respects the conditions were analogous to those found in the cases recorded in this paper. Balfour's difficulties were encountered essentially after reduction of the herniated structures. By laparotomy he found no difficulty in withdrawing the abdominal viscera from their position in the thoracic cage, but it was a struggle to prevent them from being sucked back. This was accomplished by introducing large retaining packs of gauze held in a position for retention by outspread hands. So it was in Case II: O'Connell was continually annoyed by the escape of the abdominal viscera from behind the retaining packs. Our embarrassments occurred earlier and consisted of an almost insuperable task met in passing the herniated stomach and intestine through the aperture in the diaphragm into the abdominal cavity until a vacuum was created within the abdomen as already described. Catgut was used in both of these cases for closure of the apertures. In one the continuous suture was used, and in the other the interrupted stitch was employed. With the interrupted suture, small open gaps remain, while the continuous stitch makes a tight line. With several interrupted supporting sutures this airtight closure is to be preferred.

Conclusions. 1. Early recognition of diaphragmatic hernia has reduced the surgical mortality from 75 per cent to 10 per cent.

2. An important type resulting from external violence is now recognized among pedestrian children.

3. Many congenital hernias of the diaphragm do not require operation.

4. The thoracic operation affords a direct approach; also many advantages in dealing with intrathoracic adhesions and actual repair of the aperture in the diaphragm.

5. The abdominal approach allows inspection and repair of a gap in either side of the diaphragm. Its simplicity is enhanced by a low diaphragm and the absence of adhesions within the thorax.

6. The combined operation is a third choice, preferable only to separate incisions, thoracic and abdominal.

7. The choice of operation must remain the individual surgeon's problem, to be decided upon a consideration of all the factors in each case.

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ATROPHY AND INACTIVITY OF THE PAROTID GLAND FOLLOWING OPERATIVE OBLITERATION OF STENSON'S DUCT IN CARCINOMA OF THE CHEEK

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Efforts have been made, from time to time, to arrest the activity of the parotid gland or to reëstablish by surgery the flow of saliva into the mouth for fistula of Stenson's duct.

While these efforts have been ingeniously devised and directed along experimental lines, and many important contributions, both surgical and non-surgical, have been made, the very fact of the multiplicity of the procedures seems to indicate the difficulty in selecting a method as the operation of choice.

Deaver's *Surgical Anatomy*[34] describes the duct of the parotid as: "Running about a finger's breath below the zygoma, or in a line drawn from the lower margin of the concha to a point midway between the free margin of the upper lip and the ala of the nose. It lies between the transverse facial artery above and the buccal branch of the facial nerve below. The duct runs over the masseter muscle, turning abruptly inward at its border, passes through a mass of fat overlying the buccinator muscle and beneath the facial vein and pierces the buccinator muscle, to open into the mouth opposite the crown of the second molar tooth of the upper jaw."

The etiology of salivary fistulæ is well known—how they may develop as the result of a trauma or from faulty technic in the removal of a neoplasm, the opening of an abscess or as a sequel

to flap excision in facial autoplasty. They may follow a destructive process due to carcinoma, lupus, rodent ulcer, actinomycosis or syphilis, or from a pressure necrosis and infection arising from salivary calculi.

Agnew[22] describes a congenital salivary fistula in which the opening was on the anterior part of the helix of the auricular cartilage.

Mauclaire[25] reports eleven cases of duct fistula following dental abscesses and three associated with the presence of foreign bodies, as wood and lead.

Schreiber[27] introduces a new etiologic factor, reporting a salivary fistula following a simple mastoidectomy. Such a misfortune, of course, coming from the parenchyma of the gland, demonstrates how this subject should seriously engage the attention of the operator who would deal with a tumor, phlegmon or mastoid.

An interesting factor, with a definite bearing on the end-result in the repair of a salivary fistula, has been suggested by Tait,[23] whose original work in this field has won for him distinction. The normal length of the duct of the parotid is about 6 cm. and 0.3 cm. in diameter. The oral third contains the same bacterial flora as may be found in the mouth; hence it can be definitely seen that this source of infection offers an additional factor against successful surgery, particularly if there is any oral pathology.

The possibility of the relief of a salivary fistula coming through the obliteration of Stenson's duct by ligature was first suggested by Dupuytren,[11] accepted by Velpeau and practised by Viborg and Bramann; but their efforts were far from successful, due chiefly to the development of retention salivary cystomata or infection with abscess formation.

Case Report. G. W., a man, aged fifty-eight years, was admitted to my service in the Ohio Valley General Hospital in February, 1912, suffering with what seemed to be epithelioma of the right cheek. In the center of the ulcer there was a sali-

vary fistula. Extending out from the circumference of the ulcer there was a large area of cicatricial tissue which was the seat of a dermatitis and otherwise devitalized from the over-zealous use of caustics, pastes and roentgen therapy.

Luetic infection was definitely excluded and on section the unhealthy neoplasm proved to be a cancer. There was no glandular involvement, and otherwise the patient's health was good.

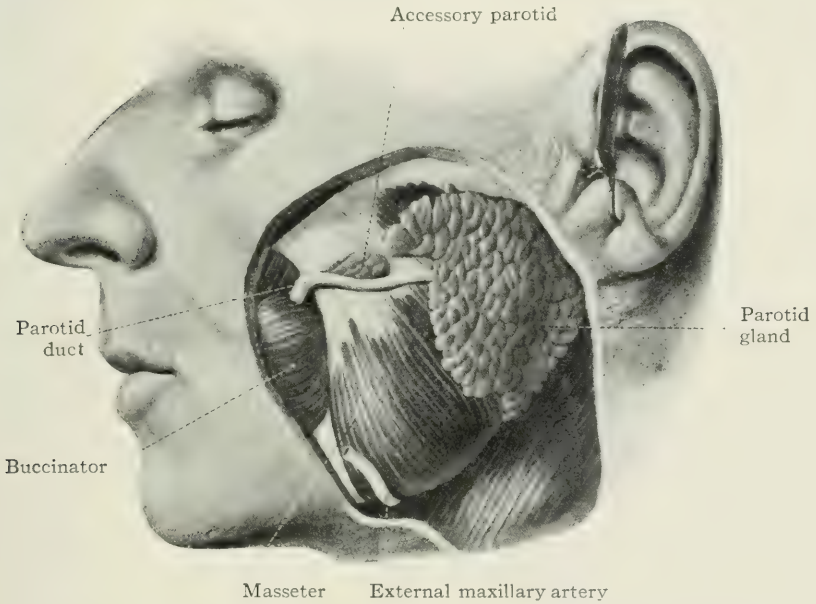

FIG. 1.—Parotid gland and Stenson's duct. (From Sabotta's Anatomy. W. B. Saunders Co.)

In planning the operation for the removal of the malignancy the question of side-tracking the secretion of the parotid presented obvious embarrassment, due to the apparent destruction of a large part of the buccal portion of Stenson's duct. Flaps were designed by horizontal H-shaped incisions, radically removing the pathology without involving the mucous membrane of the mouth. Further dissection and retraction of the posterior flap exposed the remnant of the masseteric portion of the duct,

but the short proximal end precluded the possibility of following any of the operations suggested for diverting the flow of saliva into the mouth. Ligation of the duct with the chances of leakage, infection or cyst formation, with the probable defeat of the plastic repair, was not deemed wise. The remaining part of the duct was crushed with an angiotribe as far back as possible, and this short ribbon was brought through a stab incision in the posterior flap onto the cheek and sutured to the skin.

The sole apology for this step in the technic was the hope entertained that one of two things might occur: either that the secretion of the parotid might find a new outlet far removed from the reparative work, or, however, inconsistent, a happier result might be attained in the complete suppression of the physiologic activity of the gland.

The skin wound was closed with horse-hair and provision for drainage was effected by a stab wound beneath the inferior maxilla, carrying a narrow rubber band.

The wound healed without interruption, and later the region of the parotid subjected to occasional *x*-ray treatment. There was at no time any further discharge of saliva from the obliterated duct, and there was an absence of any phenomenon of retention or painful tension on the part of the gland.

Our reason bids us assent to the accomplishment of a fact, namely, the inactivity and atrophy of a gland following the operative obliteration of its duct; but the question may be raised, did the hoped-for changes in the histology of the parotid come as a result of our aid or in spite of it?

Any further report of the progress of the carcinoma with the sequence of events is not germane to this paper; suffice to say that for almost three and a half years there was no sign of recurrence. But after that time the cicatricial tissue of the cheek, devitalized and scarred by repeated preoperative roentgen therapy and other remedial measures, offered little resistance to a rekindling of the disease. Despite the most skilfully applied *x*-ray cross-firing, radium and electrocoagulation, the cheek, bone and meninges, in slow succession, yielded to the devasta-

tion, with the inevitable *exitus lethalis* in February, 1919. There was no autopsy.

Comment on Changes in the Gland. Very little has been written of the changes occurring in the parotid gland and its secretory function following operative obliteration of Stenson's duct. It is obvious that the subject needs further experimental investigation before we can depend on atrophy or involution taking place following this procedure.

Pietri's observation leads to the assumption that atrophy comes about very slowly; this is particularly true following a non-infected fistula, while quite the contrary is the rule in cases of parotitis consequent upon traumatism, when the inactivity of the gland is rapid and permanent. In most of Pietri's cases the glandular tissue became sclerotic, adhering firmly to the tissue framework, which before had acted as a support pure and simple. But this is a hypothesis which the writer seems to think may be verified by the histologist.

In a personal communication from Frederick S. Lee, of the department of physiology of Columbia University, he found an account of some investigations by Maximow, in which in a dog the duct of the submaxillary gland and the duct of Bartholin (the chief duct of the sublingual gland) were ligated and the dog was killed after thirty-one days. The ducts on the gland side of the ligature were found enlarged and filled with thick saliva. The glands themselves were considerably diminished in size, although the interlobular connective tissue appeared strongly hypertrophied.

Microscopic investigation showed results very similar to those after section of the chorda tympani. There was apparently a paralysis and final disappearance of the mucous cells, while the serous cells, although changed, remained and continued to secrete.

Perhaps Cole's[7] suggestion may prove helpful, following the obliterative operation, in the use of radium or *x*-rays as a post-operative measure, in those exceptional cases in which the transplanting of the duct is impossible.

Dr. Burnam, of Baltimore, has described the changes in the parotid gland after radiation as striking and quite different from those occurring in the ovary. All of the salivary glands a few hours after radiation swell enormously and the patient presents the appearance of mumps. There is a cessation of saliva and consequently a drying of the secretions of the mouth, which lasts from a few days to two weeks after such treatment. This tendency to parotitis is the first change. If a tremendous amount of radiation is given there is gradual atrophy of the parenchyma of the glands; however, to accomplish this a very large treatment must be given. The parenchyma is replaced by proliferating fibrous tissue.

Review of the Literature in the Treatment of Salivary Fistula. Pietri gives a *résumé* of the various early procedure s including the compression of the gland by Maisonneuer, caustics and agglutinants and the gold plate of Malgaigne. It is needless to say that these were of little efficacy and had their day, as did also compression of the carotid and ablation of the gland. The author speaks of the work of Mollière, of Lyons, who in 1884 endeavored to cause atrophy of the parotid by the injection of phenolated oil, and how Cocchini gave preference to turpentine for the same purpose. These efforts are of interest historically, but are deserving of no comment.

Of the older operative procedures the one planned and successfully executed by De Guise, and bearing his name, has been commonly accepted as the operation of choice. It consists of the excision of the discharging sinus in the cheek and the transfixation of the buccal mucous membrane at two points with a needle carrying either silk or wire. The ligature is tied in the mouth and the seton effect with the consequent necrosis turns aside the parotid secretion in the natural direction.

This procedure is usually successful in the simple non-infected fistula, but, as so often happens, the discharge of saliva comes from a cancerous ulcer, which has destroyed a large part of the duct, and the removal of this malignancy becomes neces-

sary by radical excision before any thought can be given to the aberrant salivary flow. It is reasonable to infer that under these conditions it would be difficult to successfully employ any of the older methods, like the operations of Nicolandi, Langenbeck and Kaufman or the ingenious oroplastic technic of Braun.

Review of Experimental Work. Ferrarini's[16] work included a series of operations on dogs which was interesting. On one side simple ligature and resection of Stenson's duct was done; on the other side ligature of the duct and suture of the parotid and the submaxillary glands. After three months he dissected out Stenson's duct and injected colored gelatin into the central end. On the side where the gland had been united the gelatin passed from the parotid into the submaxillary. In dogs long operated on the inflow was so easy that the gelatin could be made to pass out of Wharton's duct and *vice versa*.

Histologic examination of several sections showed that an intimate union had taken place between the glands of the one side; on the other side the glands showed typical atrophy processes; whereas the parotid, which had been joined to the submaxillary, had preserved its form, volume and, for the most part, its structure unchanged.

The operation on man—done only on the cadaver—is described as free from difficulty. The usual section for submaxillary removal is made, being carried about 2 cm. farther upward and backward along the border of the vertical ramus of the mandible. The submaxillary capsule is incised and the gland completely mobilized. Behind the angle of the mandible the lower end of the parotid lobe is isolated, and, when necessary, the small aponeurosis between the angle of the mandible and the anterior border of the sternomastoid is divided.

The glands are then easily approximated over a wide area. A small part is removed and the cut edges sutured, the parenchyma and the capsules being joined separately.

Ferrarini expects the operation will be clinically successful in stubborn cases of duct fistula.

Tait[30] conducted three sets of experiments on dogs, cats and rabbits. The first set consisted in double ligation and resection of 1 cm. of Stenson's duct in either the inner, middle or outer third, the resected tissues being tested bacteriologically in each case; the second set consisted of ligation of the duct without resection; in the third set a double fistula was made in the outer third and then treated by double ligation and section of the duct in its inner third. From the results of these experiments he reaches the following conclusions:

"Atrophic sclerosis of the parotid gland follows obstruction of Stenson's duct.

"Its extent is in direct proportion to the degree and duration of obstruction.

"The addition of an element of infection may hasten the hyperplastic process.

"Distention of the duct may persist long after the gland has undergone almost complete fibrous atrophy.

"Fistula of Stenson's duct may be permanently cured by double ligation and section of the duct as close as possible to the gland.

"Operation on the bacterially rich outer third of Stenson's duct is much more frequently followed by fistula than are similar interventions on the inner third of the duct.

"The effects of incomplete obstruction or stenosis of the salivary ducts are frequently found in the parotid and submaxillary glands, where, by simulating malignancy, they may lead to grievous mistakes in surgical therapy."

Surgical Treatment. The articles by Dieulafe[13] and Pietri[26] in the *Annals of Otology*, December, 1918, review some of the earlier methods of treatment. The Paris *thesis* by Rousseau,[27] included in the bibliography, gives no additional data of importance.

Aigrot[1] presents an interesting discussion of the role of the auriculotemporal nerve, and a report of the anatomic studies on its distribution in the ox, horse, pig, sheep, dog and man, with a description of Leriche's procedure and the indications for its use.

Benedict[2] gives the history of a case of gland fistula caused by syphilitic abscess. The external opening was freshened and sutured, with recovery.

Berteaux[3] reports the case of a girl, aged eleven years, who had had a duct fistula for seven years because of an incompletely healed wound from a dog-bite. The duct anterior to the fistula was not viable and he concluded to lead the central portion into the mouth cavity. It was dissected out for about 1 cm. An opening was then made through the cheek and two linen threads attached to the end of the freed portion of the duct were passed through the opening. The threads were then sewed into the lips of the buccal wound. The suture was made so as to maintain the opening of the duct. The external wound healed in twelve days and the patient was discharged eighteen days after the operation, with a buccal fistula functioning perfectly.

Carwardine[5] dissected out the branches of the facial nerves which pass through the parotid. These were held up with catgut and the whole gland removed. He used small sharp scalpels. There was temporary facial paralysis, but in a couple of months there were twitchings and in four months some movement. A year afterward the patient had good control of the facial muscles and very little difference between the two sides was apparent.

Cole[6] treated two cases by an ingenious reparative operation. The general principle adopted in this operation was that "if the duct will not reach the mouth cavity the mouth cavity must be made to reach the duct." A curved incision was made with convexity down and a small flap resected upward; this revealed the duct and a small lateral hole in the duct marked the limit of patency. The duct was freed, its terminal portion resected and two very fine catgut traction sutures were passed through its walls. The mucous membrane, covered by buccinator, was then made prominent immediately in front of the masseter by means of a small swab pressed against it from inside the mouth and a small longitudinal incision was made

through it into the mouth. The masseter was nicked at its anterior border and the margins of mucous membrane stitched to the deeper margins of the wound. Through the aperture thus made the stay sutures were passed and the duct was gently pulled into the funnel-shaped extension and the extension cut off from communication with the exterior by catgut sutures. The skin was sewed up; drainage was through a small stab incision. Each stay suture through the duct was then made to take a good hold of mucous membrane inside the mouth, so that when tied the duct was secure in place. In both cases slight suppuration with discharge of saliva occurred about the tenth day, but communication with the mouth was well established and good healing occurred in a few days. Both cases were discharged cured in three weeks.

Hamilton[17] operated on a patient with a fistula of several years' standing, enlarging the opening and curetting out from a cavity the size of a hazel-nut "some starchy gelatinous secretion, together with shell-like fragments of calculous material" lining its wall.

The anterior part of the duct was not visible; the posterior part was very short.

A hemostat was forced from the cavity through the gland and the mucous membrane cut over the point of the forceps. The channel thus formed was enlarged by opening the forceps widely. A flap was cut one-half inch wide at the base just anterior to the artificial buccal opening and tapering slightly to its tip, which was posterior to the angle of the mouth. The flap was slightly less than one-eighth inch deep. The hemostat was again pushed through the channel and the tip of the flap seized and drawn into the fistula cavity, the mucous membrane being superficial. It was sutured to the gland surface by three fine silk sutures. The external margins were pared and sutured and the buccal mucous wound sutured. A rubber drain was inserted into the new opening. There was a complete cure.

Jennings[15] described a razor-cut with subsequent scar which caused occlusion of the duct and a collection of saliva in a

tumor-like lump under the skin. A cyst-like cavity at the edge of the masseter muscle was found. A bougie passed from the mouth one and a half inches up the duct which was severed. A rubber tube was inserted into the severed end and also into the central end, which opened into the cavity. A continuous chromic-gut suture was put in so as to obliterate the greater part of it; the skin wound was left open, dressed with sterile gauze and sealed with collodion. The wound healed in about two weeks. The tube was in place on the tenth day; on the eighteenth day it had disappeared. The duct was patent two months after operation.

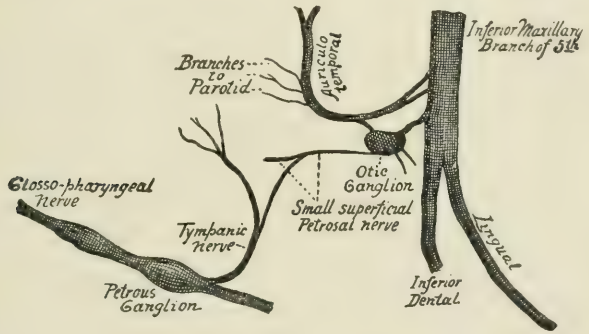

FIG. 2.—Leriche divides the auriculotemporal branch of the inferior maxillary division of the fifth cranial nerve, in his operation for fistula of Stenson's duct. (From Howell's Physiology, W. B. Saunders Co.)

Our distinguished visitor, Leriche,[20] reported cases in which he abolished the physiologic activity of the parotid by avulsion of the auriculotemporal branch of the inferior maxillary nerve. Dieulafe[14] in his endorsement of Leriche's operation mentions eleven cases in which this method was successfully employed.

The operation is comparatively simple, but it must be remembered that it does not always result in an immediate cessation of the flow of saliva. Of the cases reported in only one was the cessation immediate; in all others from two to eight days elapsed, and in most cases from five to six days, before the secretory activity of the parotid and the flow of the saliva ceased entirely.

Weitz[31] reports one case in which the fistula had persisted for more than twenty years, treated by Leriche's method. The salivary secretion ceased at once and did not reappear. He notes also that Tromp[30] had operated on three cases successfully by this method.

Cole[7] condemns Leriche's operation and likens it to the plumber who would repair the leaking pipe by cutting off the water supply of the house.

Mothersole[25] reported a patient who developed a fistula after an operation for removing a neoplasm. The amount of scar tissue surrounding the opening excluded most methods of operating, so the author dissected up a strip of mucous membrane one and one-quarter inches long and one-third inch broad, with its long axis horizontal and its front end near the mouth of the parotid duct. He then bored a hole through the cheek just in front of the fistula and dilated it so that the flap could be brought through the opening. He then dissected up the surface of the scar on the cheek and removed all but a small collar of tissue surrounding the fistulous opening above, behind and below. To this he attached the free end of the flap by a few interrupted catgut sutures. This made an epithelium lined track from the parotid gland to the mouth cavity. He dissected from the neck, just below the horizontal ramus of the jaw, a flap of skin left attached at its posterior part, rotated it through a quarter circle and covered the raw surface on the cheek with it. The gap left was closed by interrupted sutures except a small triangular bit at its widest part. The wound had healed in a fortnight and all the parotid secretion passed into the mouth. Sixteen months later a probe passed easily through the duct.

Non-surgical Treatment. Cole[6] contends that fistulæ of the gland and incomplete fistulæ of the duct tend to heal without treatment. If treatment is delayed the application of radium or *x*-rays is indicated. He writes, "Gland fistulæ and incomplete fistulæ of the duct have never failed to respond to radiations."

The radium treatment consists in exposures to a penetrating radiation from 250 mg. of radium in platinum tubes of a thickness of about ½ mm.; 3 mm. of lead were also used to cut off the hard beta rays; tubes enclosed in rubber and several layers of lint were used on the skin. Exposure of three or four hours was given each skin area, those with considerable induration of tissue receiving a longer exposure than others. In a number of patients *x*-rays were combined with the radium, small doses being given at short intervals, a 2 mm. aluminum filter being used.

The title of Kouindjy's[19] article indicated treatment of salivary fistulæ by massage and the application of hot air. The original article was not available.

Moreau[24] treated one case with silver nitrate and iodin alternately. Care was taken that the orifice was not closed too quickly. The treatment was satisfactory after a month.

Pietri[26] thinks that rather than change the course of the saliva by surgical procedure it would be more logical to try to lessen its production by absolute rest. Through the agency of what he calls a mask, which is not unlike the Barton bandage, he immobilizes the jaw, at the same time suppressing all gustatory sensation; enforces a liquid diet for several weeks, with tube-feeding, and imposes on the patient absolute silence. Thirty-eight cases were successfully treated by this method.

The original of Stropeni's[35] article is not available, but according to the abstract in the *Journal of the American Medical Association* (1920, lxxv, 138), his treatment consisted of blocking the third branch of the trigeminal nerve by injecting alcohol. This proved as efficient in curing the flow of saliva from a fistula as resection of the auriculotemporal nerve recommended by Leriche. He injected 3 cc of alcohol at the base of the skull, corresponding to the foramen ovale, and repeated this injection three days later. The fistula continued to suppurate, but there was no further secretion of saliva in the gland.

Kouindjy[36] describes his method of treating salivary fistula, as illustrated in one case of a soldier with a fracture of the

mandible, badly consolidated, and a salivary fistula of the left side of the jaw. The treatment was begun "by massage, superficial and deep rubbing, and centripetal vibrations from the periphery to the center, not touching the fistula. At the same time the facial cicatrix was massaged. All of the masso-therapeutic maneuvers were executed first without hot air, later by a current of hot air over the entire cutaneous surface of the jaw and the fistula itself. Finally the current of hot air was turned upon the buccal surface, through the opening of the mouth, in such a way as to bring it in contact with the internal border of the fistula." In order to avoid burning or pain the application of hot air was interrupted every two or three seconds. (Temperature of air, 37.9° C. to 39° C.) The treatment was begun August 20, 1918; improvement was noticeable by September 26, and the fistula was completely obliterated by December 17.

The action of massage and hot air on the fistula Kouindjy explains as follows: The hot air causes a hyperemia that induces a local change that may be compared to a caustic action. In addition it has an analgesic action on the tissues and causes progressive drying of the secretions. This last effect can be compared to resection of the auriculotemporal nerve. The action of massage is mechanical and reflex. The mechanical action has a favorable effect on the circulation and the reflex action on the terminal nerves. It also increases the nutrition of the tissues.

From this study and out of the abundance of an interesting literature, including experimental, surgical and non-surgical, we may briefly conclude:

1. That by crushing the duct of the parotid there may follow histologic changes leading to an atrophic sclerosis with suspension of the activity of the gland.

2. It is fair to assume that this condition is influenced by the use of radium alone or combined with the *x*-rays, resulting in a replacement of the gland by a proliferating fibrous tissue.

3. Further experimental evidence is wanting before we may

depend on operative obliteration of the parotid duct to arrest the flow from a salivary fistula.

4. Fistula of Stenson's duct has been permanently cured by double ligation and section of the duct as close to the gland as possible. (Tait.)

5. Leriche and his followers have arrested the physiologic activity of the parotid by avulsion of the auriculotemporal branch of the inferior maxillary nerve.

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DISCUSSION ON THE PAPERS OF DRS. ARMSTRONG, LILIENTHAL, TRUESDALE AND HUPP

Dr. John A. Hartwell, New York:

In this connection I should like to report a case operated on by Dr. Worcester, at the Bellevue Hospital for which there was no explanation of the condition found. It was a sliding inguinal hernia of the ileum, with total absence of the mesentery. At that time I had the literature looked up for an explanation. It did not give the impression of a separation of the mesenteric leaves from the ileum, but an actual absence of any peritoneal covering over the vascular portion of the ileum. Apparently this ileum, such as described by Dr. Armstrong, had slid down in the way that the large intestine may slide when it comes down in the hernia.

A rather cursory search of the literature by Dr. Coley, who was writing on the subject of hernia at that time, failed to find a description of a hernia of that type, and it was suggested that there was a congenital absence of the mesentery, but no one with whom I discussed it was able to give me any information and the literature failed to reveal any such condition.

Dr. Willy Meyer, New York:

I am sure everyone here will rejoice with Dr. Lilienthal in the recovery of his patient. I wish to offer him again my heartiest congratulations on the splendid result obtained. This case represents the second case of resection of the thoracic portion of the esophagus with recovery; the first, Dr. Torek's case, done transpleurally at the Lenox Hill Hospital, of New York, nine years ago, is still alive and in good health, evidently cured of her trouble.

As Dr. Lilienthal mentioned, the approach to the posterior mediastinum by posterior mediastinotomy has been done by various surgeons for various diseases before. In addition to those referred to by him I should like to mention the late Dr. Joseph Bryant, of New York, who wrote an interesting essay on this operation.

In the way Dr. Lilienthal has made his approach he has succeeded in giving both hands of the operator easy play so that he can thoroughly handle the diseased esophagus from behind, certainly a most important point. Still, I feel that we should not give up the transpleural resection completely. All possible ways of exposing and resecting the esophagus should be worked out. The principal question in doing transpleural resection, which gives such thorough exposure, is proper postoperative drainage. We are learning gradually how to do this.

Certainly, it will be wise to be prepared for an injury of the pleura also in the posterior approach. Dr. Lilienthal in his case was so fortunate as to be able to peel off the pleura from the esophagus uninjured.

In the course of time, if one had a series of cases for operation at disposal, I personally feel that one might try in some cases to combine the resection of the carcinoma by means of posterior mediastinotomy with antethoracic subcutaneous transposition of the proximal esophageal stump. After all, the principal work in esophagoplasty has so far been done in front of the chest. Fourteen cases of total esophagoplasty have been reported, particularly by surgeons abroad.

No doubt Dr. Lilienthal's bold and wonderful work will greatly help to push this chapter of thoracic surgery forward and to awaken the operative work for cancer of the esophagus from its long lethargic radium sleep.

Regarding Dr. Truesdale's presentation, it stands to reason that if we open the thorax we can see what we are doing in reducing the hernia. His case is certainly most interesting as showing how it became possible by lifting the diaphragmatic aperture to replace the hernial contents. I am sure in some cases, in which large portions of the abdominal contents have slipped into the thorax, that it will be advisable to combine the thoracic route with an abdominal incision in order to be sure that all the organs replaced into the abdomen are in their proper position after the hole in the diaphragm has been closed.

Dr. William A. Downes, New York:

I think, as Dr. Truesdale has said, that the question of approach in these diaphragmatic hernias should be determined in each individual case. Certainly, in traumatic cases the thing to be desired is to reduce the contents into the abdominal cavity, but in the case which I reported of undoubted congenital origin it was necessary to overcome the obstruction at the pylorus by performing a gastroenterostomy through the dilated esophageal opening, as it was impossible to draw the stomach into the abdominal cavity. That the stomach may remain in the chest and function perfectly is shown by a case of ours, aged sixty years, in which the *x*-ray taken for another purpose showed the entire stomach to be above the diaphragm.

Dr. Alexis V. Moschcowitz, New York:

I have followed Dr. Lilienthal's very successful case with so much enthusiasm and interest that I am truly envious. I have on numerous occasions expressed myself as wishing that I had done it.

Dr. Howard Lilienthal, New York (in closing):

I mentioned Dr. Bryant and I mentioned everybody that Dr. S. Hirshfeld, of New York, who looked up the references for me, found in that literature.

Dr. Bryant did not do work on the esophagus but did work on the approach to the mediastinum.

As to Dr. Meyer's suggestion of continuing the transpleural operation, I will say that attempts have been made hundreds of times, and every patient has died. If Dr. Meyer still wishes to continue the transpleural method I shall be glad to note his success.

Also, I should like to call attention to something which I did not mention in this brief abstract of my paper. *No gastrostomy was done.* I do not advise that. If one is going to do this operation I think, as a rule, gastrostomy should be done, and in any other cases which I may have I shall probably perform a preliminary gastrostomy, but only for convenience and not on account of necessity. The patient on whom I operated was still able to swallow fluids, and for the two weeks between the two stages of the operation he was nourished sufficiently well because his stricture had been stretched by the stomach tube at the time of the operation.

As to the subcutaneous methods of esophagogastrostomy for non-malignant stricture they are very difficult and require numerous operations. I think in one case sixteen operations were done by Dr. Kirschner, who finally succeeded, but without resecting the diseased esophagus. I do not believe that method should be used if one can possibly avoid it. Most of the cicatricial strictures can be dilated.

A word about Dr. Truesdale's remarkable case: Before he read his paper I believed in the transthoracic operation in these traumatic cases, but after seeing the diagrams and the *x*-rays I have come to the conclusion that in those cases of prolapse of abdominal viscera into the chest it would be better to operate from below, because one has the diaphragm in plain view and within easy reach. If afterward the transpleural operation is required it can be done. This condition is different from gunshot wounds involving the chest and diaphragm, in which I am convinced the approach should be from above.

I do not think we should call these cases hernias, for they are not: they are prolapse of the intestine through an injury in the diaphragm. As Dr. Truesdale stated, there was no sac, and the classical definition of a hernia would include the presence of a sac.

Dr. P. E. Truesdale, Fall River, Mass. (in closing):

I may say that these little patients are usually seen first by the surgeon, and the recognition of the condition is very essential. The

key to the diagnosis may be observed on inspection. In both children which I examined there was a distinct outward flaring of the left costal margin. That is due to the paralysis of the diaphragm and the action of the transversalis and scalene muscles.

Dr. Vilray P. Blair, St. Louis, Mo.:

Naturally I could claim little to add to such a presentation of a subject that is not often brought to the attention of surgeons, and yet it seems to me a pity to let this presentation go without comment.

M. le Dr. Mauristan treated his fistula by ligation, and as far as I know had no trouble from it. I have had occasion, in carcinoma of the cheek, to cut across the duct quite a number of times with the cautery, leaving the duct so that the saliva could drain into the mouth; but it is my impression that the gland was put out of business in every instance. Also in our dissection of the neck for carcinoma we remove the lower half of the gland. I have seen two cases of fistula behind the ear following abscess and I have seen but one of our own cases in which a fistula persisted. I am therefore of the opinion that scar pressure will cause atrophy in most cases where the duct has been cut across.

Suppression of the secretion may be a desirable thing under other circumstances. After removal of a stone from the parotid gland, and even from the parotid duct, one is liable to have a constriction in the duct that keeps up symptoms for a long time, and in such cases it would be desirable to do away with secretion.

The ordinary interpretation of a ranula is that it is due to an obstruction in the flow of saliva from the sublingual gland. Why you should get a cessation of flow from an obstruction in a parotid duct, while in a sublingual it should persist and form a cyst, has not been explained.

THE SIMPLIFICATION OF TECHNIC IN OPERATIONS FOR HARELIP AND CLEFT PALATE

By JAMES E. THOMPSON, M.D.
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The object of the following communication is to explain in a concise manner the anatomical faults present in harelip and cleft palate and to formulate rules of technic by which the deformed structures can be brought into the position they would have occupied if union of the component parts of the face and palate had occurred at the proper time, that is about the seventh week of intrauterine life.

We have never been able to explain why the edges of the embryonic fissures of the face and palate fail to unite. There is no reason to believe that the failure is due to lack of development of the lateral halves of the palate or of the alveolar border, because in almost all cases careful measurements taken soon after birth show that the superficial area of the lateral processes is quite sufficient to complete the roof of the mouth, if the maxillæ could be approximated and the palatal plates changed from their vertical to a horizontal position.

The moment embryonic fusion fails, the great and probably the only agent in preventing subsequent union is the tongue, which exerts constant pressure between the edges of the cleft until it succeeds not only in prying the jaws apart but in pushing the palatal plates upward until they come to occupy a somewhat vertical position. The mechanism of this disrupting force can be clearly traced in every case. Whenever the gap is large enough for the tongue to be inserted, evidences of muscular

pressure are always present and the direction in which the disrupted parts are moved is always that of the tongue thrust, *i. e.*, from behind forward and from below upward. Since the force exerted by the tongue is greatest at the tip and decreases gradually as we pass backward toward the epiglottis, where it reaches its minimum, we should expect to find the deformity greatest where the prying force of the tongue can be exerted to greatest advantage. The expectation is supported by the actual findings. Thus, in clefts confined to the soft palate the muscular action of the posterior part of the tongue is so feeble and so ineffective as to be negligible. The slight disrupting force exerted is more than neutralized by the mobility of the muscular halves of the velum. The deformity is consequently very slight. On the other hand, in clefts extending through both soft and hard palates but stopping short at the alveolar margin, the tongue exerts considerable force on the lateral palatal plates of the maxillæ, forcing them upward and outward against the lateral walls of the nose and producing an unusually wide cleft. In addition, the anterior and lateral portions of the alveolar ring are also pushed outward to a considerable extent. But the severest degrees of deformity occur in examples of complete palatal cleft extending through the alveolar margin into the nostril. The tongue has full play and acts like a wedge, driving the maxillæ apart in front so that they swing outward like double doors on hinges which are situated behind near the posterior ends of the alveolar borders.

The principles of treatment are founded on accurate knowledge of the anatomy of the deformity; in other words, on a correct estimate of the degree of distortion. This can be measured graphically by comparing the deformed alveolar border with one reconstructed from a mold taken of the alveolar border of the mandible of the patient. In using mandibular measurements as a standard we must not forget that under normal conditions the articulation of the mandible is a little outside that of the maxilla. Therefore the maxillæ must be molded until the alveolar ring is a little smaller than that of the mandible.

For our purpose it will simplify matters to base the principles of treatment on an analysis of the anatomical features of a few selected common types of deformity. In this way we shall avoid unnecessary detail. To this end we have selected three types:

I. Complete unilateral cleft of the lip, unilateral cleft of the alveolar border and complete cleft of the palate.

II. Complete bilateral cleft of the lip, bilateral cleft of the alveolar border and complete cleft of the palate.

III. Intact lip, intact alveolar border, complete cleft of the palate.

TYPE I.—COMPLETE UNILATERAL HARELIP ASSOCIATED WITH A CLEFT EXTENDING THROUGH THE ALVEOLAR BORDER OF THE JAW AND THROUGH BOTH HARD AND SOFT PALATES

Fig. 1 illustrates the text. It is a life-size tracing of a plaster model of a typical case of this variety of cleft.

The points marked *B* and *B'* represent the anterior ends of the alveolar borders of the maxillæ and are placed on parts that would be in contact if the jaws had united. The distance *B* to *B'* varies within great limits. It is often as much as 15 mm. across. The points marked *A* and *A'* are on the alveolar border at the site of their most pronounced lateral bend. The distance *A* to *A'* indicates the greatest transverse measurement and it is always greater than it would be if the maxillæ had united. By comparing it with the corresponding measurement across the mandible an accurate estimate can be formed as to the distance the two halves of the upper jaw have been thrust apart. We have found in average cases that the measurement is several millimeters greater than it would be in a normal palate. The points *C* and *C'* mark the posterior end of the alveolar ridge. For all practical purposes this indicates the locality where the maxilla and palate bones are connected. The *X* represents the position of the hamular process of the pterygoid. The distance CC' is seldom increased materially. On the average the increase will

not be more than 1 or 2 mm. The side which carries the premaxilla (the larger one marked *E* in the figure) always receives the full force of the tongue thrust, and in consequence its anterior end *B* is pushed outward and forward. The smaller side, *E'*, receives very little tongue thrust and consequently its position is changed very slightly. As a rule, its alveolar border articulates fairly accurately with that of the mandible.

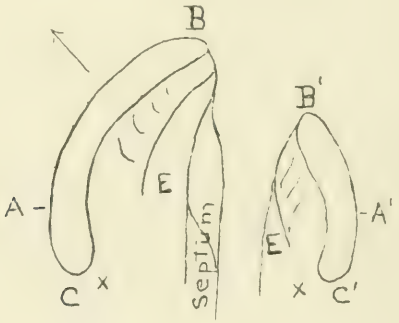

FIG. 1.—Life-sized sketch of a plaster mold of the palatal surface of the maxillary plates of a case of unilateral cleft of the alveolar border and complete cleft of the palate. The letter *E* is placed on the right maxillary plate, to which septum and premaxilla are attached. The letters *A*, *B* and *C* refer to points on the alveolar border, which are described in the text. The cross is placed over the hamular process. The arrow shows the direction in which the larger plate is forced outward by the tongue thrust.

The lateral palatal plates deserve careful scrutiny. The mucous membrane covering each plate consists of two distinct areas: A lateral, which forms a strip running along the alveolar border, which is thick, rough and corrugated; and a median, bordering on the cleft, which is thin and smooth. The larger lateral plate, *E*, usually fuses with the septum. When the septal attachment is complete the nasal cavity on this side is closed off as far back as the junction of the hard and soft palates. It is usually well formed and roomy; but in some instances where tongue pressure has been excessive both lateral palatal process

and septum are crowded outward, the result being that the lower portion of the septum is bent into a somewhat horizontal position near its lower attachment and the nasal passage on this side is considerably narrowed. The smaller lateral plate, *E'* has no attachment to the septum. As a rule, it lies somewhat vertically, with its free edge pointing upward, having been pushed upward by tongue pressure. A study of Fig. 2, *A* and *B* (a coronal section of upper and lower jaws of a seven-months' fetus with cleft palate), shows most of the points emphasized in the above description. The effects of the tongue thrust on the lateral palatal plates and septum are particularly evident. The smaller palatal plate is almost vertical and its free edge is opposite the septum at a point fully 2 mm. higher than the attachment of that process to the other palatal plate. Its cranial surface has been squeezed against the turbinated bones of this side. The larger lateral plate is pushed upward to a slight extent only, because the septum has acted as a buttress to protect it from pressure. The lower portion of the septum has been molded into a somewhat horizontal or slanting position.

After a careful study of a large number of models of this type of deformity the following conclusions seem to be justified:

1. If embryonic union fails, the resulting deformity seen at birth can be accounted for in every detail by the muscular action of the tongue.

2. That there is little or no separation of the posterior ends of the maxillæ from one another, and that any movement here is in the nature of a hinge movement by which the posterior end of the maxilla and the vertical plate of the palate bone swing on their attachments to the pterygoid processes of the sphenoid.

3. That the side to which the premaxilla and the septum are attached (the larger side) is more affected by the tongue thrust than the other maxilla, the result being that its anterior end, *B*, swings outward and forward on its hinge (situated behind at *C*), taking with it the septum and nose and carrying the alveolar border to a plane far outside and in front of its normal position; and further, that the attachment of the septum to the palatal

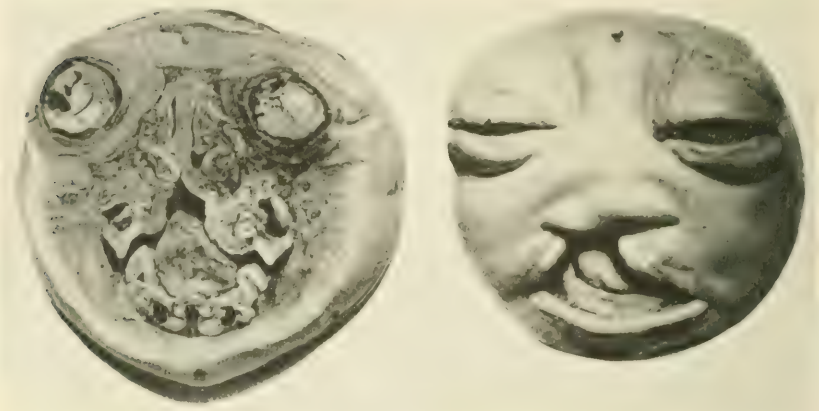

FIG. 2.—*A* shows photographs of the head of a seven months' fetus in which a complete cleft of the lip, alveolar border and palate was present. On the right, the external appearance of the flaring nostril and the cleft in the lip are well depicted. The tongue and alveolar margin of the mandible stand out in relief. On the left, we see a coronal section of the face passing through the middle of the palate. The anatomical features of both photographs are emphasized and labelled accurately in Fig. 2*B*.

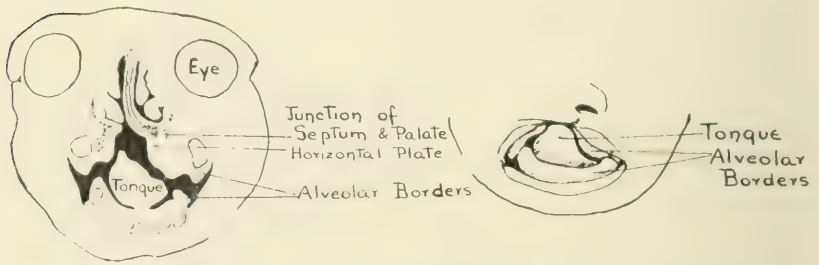

FIG. 2*B*.—Drawing of previous figures to show the exact relations of the palatal plates to one another and the exact boundaries of the cleft. The site of fusion of left palatal plate and septums is indicated by an acute bend. The lower part of the septum is bent like a hockey stick. Both palatal plates have been pushed into a vertical position. The free edge of the right palatal plate (left in the figure) is considerably higher than that of the left. The alveolar border of each maxillary process lies outside that of the mandible. Under normal conditions the alveolar border of the mandible should lie a little outside that of the maxilla. The tongue occupied the space between the separated maxillæ. The apex of the triangular-shaped section was placed between the edge of the left maxillary plate and septum.

plate has enabled it to withstand the vertical thrust of the tongue and to retain a reasonably good horizontal position.

4. That the other maxilla (smaller side) is displaced as a whole very slightly in a lateral direction, but that its horizontal palatal plate is seriously deformed, being frequently thrust upward into a vertical position against the turbinate bones.

It is evident from the preceding description that the exact measure of the separation of the maxillæ from one another can be obtained from measurements taken between corresponding points on opposite sides of their alveolar borders and comparing them with corresponding measurements taken across the semicircle of the alveolar border of the mandible. We have found that the measurement CC' is seldom greater than a similar measurement across the mandible. In an average case the measurement AA' is usually 2 mm. or at the most 3 mm. greater than normal. The distance BB' varies within great limits. It is often 10 mm. and sometimes 15 mm. in length. The statement made by Brophy[1] and others, that the width of the cleft is the true measurement of the extent of separation of the maxillæ, is not supported by facts. It can be true only in instances (very rare) in which the lateral maxillary processes have retained their normal horizontal position. When the lateral processes are in a vertical position (the great majority) the edges of the cleft are carried with them and this factor increases the width of the cleft. As a matter of fact, the true estimate of the width of the cleft is obtained by taking the sum of two displacements: (1) The lateral displacement of each maxilla, and (2) the vertical deviation or slant of the lateral palatal plate. From

[1] "I have found that the upper jaw in comparison with the lower is just as much broader than it should be as is the distance between the borders of the tissues." *Brophy's Oral Surgery*, p. 580.

"The upper jaw is just as much wider than it should be as the distance between the borders of the cleft." *Brophy's Oral Surgery*, p. 593.

"In complete cleft palate the maxillary bones have failed to coalesce and are separated from each other by the width of the cleft. Theoretically, therefore, the approximation of these bones should bring together the edges of the cleft." Berry and Legg, *Harelip and Cleft Palate*, p. 261.

descriptions of Brophy's operations the impression is given that the edges of the cleft can be brought into contact by merely approximating the sides of the maxillæ. A glance at Fig. 2 (left) will show the fallacy of this idea. Approximation of the maxillæ would bring the edge of the smaller right, vertical palatal process against the side of the septum, but could never bring it in contact with the edge of the opposite palatal process. Contact of the edges of the cleft can be secured by mere approximation under two conditions only: (1) In the rare cases where the palatal plates have retained the normal horizontal position, and (2) in cases where the palatal plates have been thrust symmetrically apart. The latter condition is seen in most cases of complete cleft palate with a free septum. A little reflection, however, will convince us that approximation in the latter condition would be inadvisable, and if persisted in would result in serious disfigurement, because the jaw would be narrowed to such a degree that a long, narrow beak would result.

TYPE II.—COMPLETE DOUBLE HARELIP; BILATERAL CLEFT THROUGH THE ALVEOLAR BORDER; COMPLETE MEDIAN CLEFT THROUGH BOTH HARD AND SOFT PALATES

Fig. 9 is a life-size sketch of a plaster mold of a typical example of this deformity. The septum is massive. It hangs downward like a ploughshare and is unattached to either lateral maxillary plate. It shows a bend to the right along its whole length. Its anterior end is elongated and projects in front of the anterior ends of the maxillæ. The premaxilla is attached to the front of the septum. It forms a massive projection, distinctly divided into two prominent globular swellings which contain the unerupted central incisor teeth. A small erupted tooth, which represents an accessory incisor, is shown attached to the lateral aspect of the right globular process. (The position of the premaxilla and the unusual length of the septum suggests excessive growth. In reality, the increased length of the septum is produced by the molding and stretching of the soft bone at its

anterior end by the pressure of the tongue on the globular process. Not infrequently this process assumes a horizontal position, and seems to project from the tip of the nose, the columella being almost obliterated.) Both maxillary processes are symmetrically placed as to each other, but careful scrutiny shows that the left process has suffered more from tongue pressure than the right, in that the anterior end of the alveolar process has been pushed a little further outward and the palatal plate a little higher. The *right maxillary process*, *E*, is separated from the globular process and the anterior end of the septum by a narrow fissure about 1 mm. wide. Its alveolar margin curves around in front of the palatal plate and forms the margin of the fissure between it and the globular process. The palatal plate is well developed and shows little upward displacement. The rugose and smooth areas of mucous membrane are normally distributed. The *left maxillary process*, *E*, is separated in front from the globular process by a distance of 3 mm. and from the edge of the septum by a distance varying from 5 to 7 mm. The alveolar border does not curve around the front of the palatal plate. The palatal plate occupies a higher level than that of the right side. On careful inspection it is found that the part of the palatal plate covered by rugose mucous membrane lies in its normal horizontal position, while that covered by smooth mucous membrane is vertically placed. The free edge of the cleft on this side is almost 2 mm. higher than that on the right. It is evident, here, that the tongue has exerted its greatest pressure in the angle formed by the junction of septum and premaxilla and in the space between these parts and the front of the left maxillary process, and that the effect has been to thrust the maxillæ apart and to force the premaxilla forward. The measurement *CC'*, taken across the space between the posterior ends of the alveolar margin, was about 3 mm. greater than that across the ends of the mandible. The other measurements, *AA'* and *BB'*, were more difficult to fix accurately, because it was next to impossible to indicate with absolute certainty corresponding points on maxilla and mandible.

By comparing outlines of the superimposed molds of the alveolar border of upper and lower jaw (Fig. 10), the extent of the deformity is shown in a graphic manner. From this we computed that the measurement AA' was 9 mm. and CC' 3 mm. greater than normal. For practical purposes we can assume that the maxillæ have been pushed apart a distance of 3 mm. behind, at CC', a distance of 9 mm. in the middle at AA', and at least 9 mm. in front at BB'. (The actual measurements of this specimen are described in detail on page 374.)

TYPE III.—COMPLETE CLEFT OF THE PALATE WITH INTACT ALVEOLAR BORDER AND LIP

Fig. 13 is a life-sized sketch of a plaster mold of a typical example of this deformity. The alveolar border is frequently well formed and seems apparently undeformed. If, however, careful measurements are taken and compared with those of the lower jaw, it will be found that the greater part of the circumference of the alveolar ring of the upper jaw is expanded. The expansion affects the middle and anterior parts of the semicircle almost entirely. As a result the lower jaw articulates well within the ring of the upper and the chin recedes. The palatal plates are often pushed into an exaggerated vertical position, and, as a result, the edges of the cleft are placed high up in the nasal cavity and the cleft is unusually wide. This is more evident in cases where the cleft extends as far forward as the premaxilla. In the case figured the whole length of vomer stands out in relief and hangs free in the middle of the cleft. On either side of it there is a deep recess, the lateral walls of which are formed by the palatal plates of the maxillæ which have been thrust into a vertical position. The smooth area of mucous membrane is not shown in the drawing because it was out of perspective. The rugose area is well in view.

It is not necessary for our purpose to describe the anatomical features of all the numerous varieties of fissure in the lip which are associated with the types of bony deformity which we have

just described. Most of the standard operations commonly used will give excellent cosmetic results if attention is paid to the basic principles of plastic surgery and the bony deformity is corrected. We must, however, insist that correction of the bony deformity ought to precede every operation, because it is impossible to repair a lip that will satisfy critical inspection unless we have previously restored the curve of the alveolar border of the jaw, straightened the nasal septum and refashioned the anterior nasal aperture. The only way to remedy a flattened, slit-like ala nasi is to straighten the nose by restoring

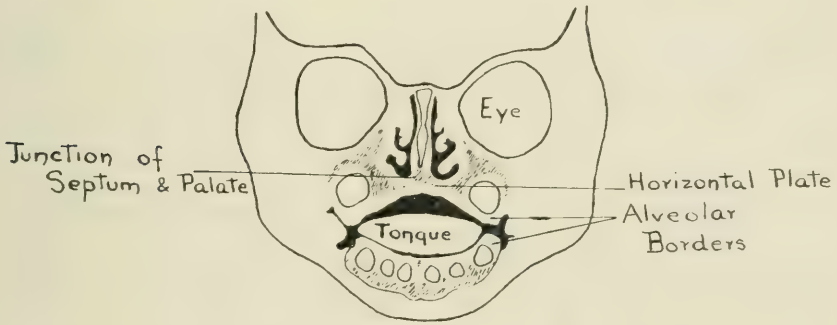

FIG. 3.—A drawing of a coronal section of the face of a normal fetus at term. The palatal plates and septum have united. Note the shape of the tongue molded to the normal palatal arch. The alveolar border of maxilla and mandible articulate with each other accurately on the left side. On the right, that of the maxilla is slightly outside the alveolar border of the mandible.

the pyriform aperture of the nasal cavity. A full prominent lip can be obtained only by restoring the curve of the alveolar border, which then serves as a cushion for the lip to rest upon.

Once the bony deformities are remedied the repair of the lip and nostril is a matter of detail, the principles of which can be enunciated under the following heads: (1) The fashioning of the nostrils; (2) the making of a lip of proper depth; (3) restoration of the vermilion border and the red line of the lip.

1. *The Fashioning of the Nostrils.* Developmentally, the margin of the nostril is formed by the union of the lateral and medial

nasal processes, both of which belong to the premaxilla. Cases of harelip are occasionally seen where the lip is cleft almost into the nostril, but the ring of the nostril is completed below by a narrow strip of skin of a triangular shape attached by its apex to the side of the columella. Fig. 4, *B*, shows this developmental abnormality. In Fig. 4, *A*, we see the lines of embryonic fusion along which defects may occur. Clefts in the lip proper are always due to failure of union between the maxilla and premaxilla. If we examine carefully the margin of the

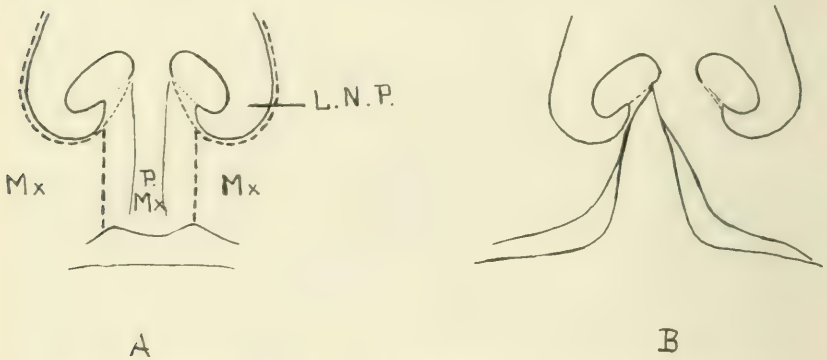

FIG. 4.—The figures show in the lip and nostril the lines of fusion of maxillæ and premaxilla, which is emphasized by the dotted red line. The ring of the nostril is completed below by the fusion of external and internal nasal processes. This is indicated in *A* by a triangular area bounded by black dots. In *B* an example of incomplete harelip is shown, in which the margin of the nostril is intact, the lower part being formed by the external nasal process.

nostril in a case of complete cleft we will find the following appearances. On the surface of the lip, just medial to the projection which forms the ala nasi, there is a smooth area of lip which ends abruptly in a hump which forms the corner between the cleft and the nostril margin. This surface represents the triangular strip which belongs to the lateral nasal process. The hump represents the point which should be attached under normal conditions to the side of the columella. On the medial side of the cleft, extending from the columella, there is a similar smooth

area which occasionally (but more rarely) shows a similar hump or projection. If the hump on the outer horn of the nostril be brought in contact with that (or the area) on the inner horn, it will be found that the ring of the nostril will be restored. If a measurement is taken of the circumference of the sound nostril and that of the nostril restored in this manner, they will usually coincide. If there is any difference it will be caused by the difficulty in fixing the point on the median side (columella) to which the projection on the lateral side is opposed, because quite frequently the projection is wanting on the former. If there is no projection on the median side of the columella, we bring the projection on the lateral corner of the cleft over to the side of the columella until the circumference of the nostril is restored accurately. We then fix this point indelibly by transfixing it with a horsehair stitch. We then have the two corners of the horn fixed, and if care is taken not to shave them when the sides of the cleft are being pared, the restoration of the nostril will be perfect. If these details are carried out scrupulously the ala nasi will be carried into the position it ought to occupy, *i. e.*, it will be carried medially and upward, and its natural curve restored. Careful comparison with the sound nostril is necessary.

2. *Making a Lip of the Proper Depth.* In cases of single harelip the exact depth of the lip required can be determined accurately by measuring on the sound side, directly downward from the lower margin of the nostril to the mucocutaneous margin. Screw compasses with sharp points are used for this purpose. We then measure the same distance from the hump or projections (or suture) previously fixed at the margin of the nostril, to the mucocutaneous junction on either side of the cleft and fix the place indelibly by a prick of the point of the compasses. This measurement fixes the length of the lateral incisions required to make the lip deep enough when the sides of the cleft are pared and brought together.

3. *Restoration of the Vermilion Border of the Lip so that the Mucocutaneous Junction Runs Continuously from Side to Side*

Without a Break and the Mucous Membrane is of the Proper Depth. Approximation of the sites of the punctures made at the mucocutaneous junction by the points of the compasses after the edges of the cleft have been pared will restore the red line of the lip accurately. Proper depth of mucous membrane is obtained by measuring from the mucocutaneous puncture to a point on the free margin of the lip near the corner of the cleft, a distance a little greater than the vertical depth of the mucous membrane immediately below the puncture. This point is fixed by pricking with the compasses. A similar point is fixed on the opposite side of the cleft. We have then fixed definitely by measurements three points on each side of the cleft, one at the upper corner of the cleft, another at the mucocutaneous junction and the third at the free edge of the lip. The lip is now transfixed by a cataract knife at the point on the mucocutaneous junction. From this point it is carried upward with a slight outward curve and cuts its way into the cleft just median to the hump (or puncture) at the margin of the nostril. The mucous membrane of the lip is then divided by an incision which passes from the puncture on its free margin to the point of transfixion. The procedure is then repeated on the opposite side of the cleft. This produces two lines of incision, apposed to one another, that will coincide accurately if the points previously marked are brought into contact. The steps of this procedure are shown in Fig. 5 (1, 2, 3 and 4).

The foregoing description applies to cases of single harelip. Double harelip requires special consideration. The area of lip attached to the premaxilla, which under normal conditions forms the philtrum, is usually very small. Quite frequently it forms a small projection with a free semicircular border attached to the end of a short, poorly formed columella. It appears to stick out from the end of the nose. It can rarely be used, as was the developmental intent, to make the whole depth of the new lip, because it is too short to cover the alveolar border of the premaxilla, much less the incisor teeth after eruption. It is therefore used to form the upper medial portion only of the

new lip. The lower part is formed by bringing the lateral (maxillary) portions of the lip together in the middle line below the philtrum. From an embryological point of view the method is imperfect, because the central portion (or philtrum) of the

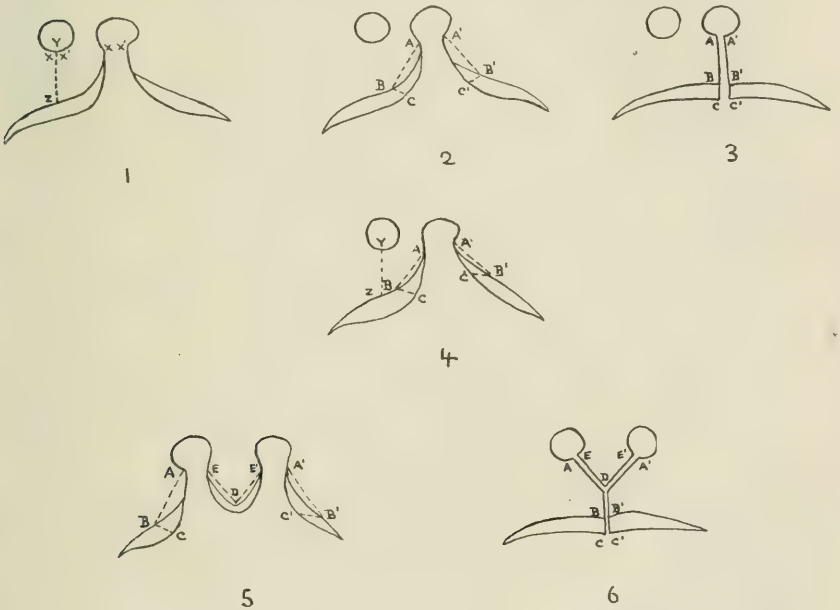

Fig. 5. The numbers 1, 2, 3 and 4 illustrate the method employed in operating on single harelip. The measurement *Y Z* in No. 1 determines the depth of the lip. In planning the incision the point *A* is placed on the margin of the nostril, *B* is at the mucocutaneous junction, and *C* is on the free margin of the lip. *A'*, *B'* and *C'* are similar points on the opposite side of the cleft. The distance *A B* is made equal to *Y Z*. The distance *B C* is a little greater than the shortest depth of the mucocutaneous margin. The distance *A B* — *B' C'*. No. 3 shows the result when the edges of the incision are brought together preparatory to suture. No. 4 is an example of a cleft with widely divergent sides and irregular depth of mucocutaneous margin. It brings out the irregular direction of the incisions required to secure accurate coaptation. In the numbers 5 and 6, the steps of the operation used in double harelip are shown. The diagrams are self-explanatory. The distance *A B*, which determines the depth of the lip, is more or less arbitrary. It is usually obtained by drawing a line across the cleft with a probe bent to represent the repaired lip and dropping a perpendicular to this line from the coapted margins of the nostril *A* and *E*.

new lip is not obtained exclusively from the premaxillary covering. The usual way in which the soft parts are approximated is the following: The premaxillary lip is carefully pared either into a triangular or semicircular form by removing all the mucous membrane from nasal corner to nasal corner. Then the lateral margins of the cleft are pared according to the rules previously laid down. Care is taken that the distance AB must always be greater than the distance ED, otherwise when the parts are brought together the point D would lie between B and B', and would break the line of the vermilion border of the lip. It is very seldom that the central part of the lip is deep enough to necessitate such precaution. The steps of the procedure are shown in Fig. 5 (5 and 6).[1]

Now we are in a position to trace step by step the operative procedures required to restore the jaws, lip and palate as closely as possible to their normal state.

For many years it has been my aim to finish the work at as early an age as possible, because I felt sure that the chances of normal speech would be favored if the palate could be closed before the child learned how to talk. It has been my custom for many years to operate on the lip and alveolar border as soon after birth as possible. I have postponed the operation on the palate to a much later date for two reasons: (1) Because very early operations on the palate are attended by a high mortality, and (2) because the operation is easier to perform when the baby is older. After the age of six months most well-nourished babies are strong enough to undergo palate operations, and there is comparatively little danger to life if the operations are short in duration and attended by slight loss of blood. In one case of unilateral complete cleft of the lip, associated with cleft of the alveolar border and complete cleft of the palate, repair of lip and palate was completed when the baby was five months old. This is the earliest data in my series (Case II, J. H., second alternative). The next earliest case was six

[1] The above description is a modification of that previously described by the author in *Surgery, Gynecology* and *Obstetrics*, May, 1912, p. 498 to 505.

months old at the time of the operation on the palate (Case I, M. B., first alternative). Both were favorable cases. The babies were exceptionally robust and the clefts were narrow. In the great majority of cases we are fortunate if we can complete the closure of the palate before the child begins to talk. Even under the most favorable conditions, for one reason or another, the babies will be a year or eighteen months old before the work can be completed. Double harelip associated with complete cleft of the alveolar border and complete cleft of the palate stands in a class by itself, and complete closure of these palates is rarely possible before the end of the second year.

The types of deformity described previously will now be taken in order and each stage of the reconstructive process taken up step by step.

TYPE I.—COMPLETE UNILATERAL HARELIP, UNILATERAL CLEFT OF THE ALVEOLAR BORDER AND COMPLETE CLEFT OF THE HARD AND SOFT PALATES

Occasionally, when the cleft is narrow, reconstruction can be completed in two stages. Usually three operations are needed and in a few cases four. Each case is a law unto itself. The sequence of the operative stages is determined by the nature of the first operation and the extent of reconstruction accomplished at this time. The attached table shows in a graphic manner the complete dependence of each subsequent operation on the first. It is an accurate mirror of my own practice. In the text the scheme will be followed as closely as possible.

In my earlier work I followed the operative sequence given in the "first alternative," and found that the results were very satisfactory. There was, however, one serious objection. In the second operation it was very difficult to repair the extreme anterior end of the cleft in the hard palate, and as a result a small opening persisted that was very hard to close. This led us to try the "second alternative," in which we were ambitious enough to try to repair alveolar border, anterior part of hard

palate and lip in one sitting. We found that the results were satisfactory and that the palate could be closed subsequently in one operation. Unfortunately, the operation was followed by severe shock and was unsafe unless the baby was unusually well-nourished and robust. This led us to employ the "third alternative," in which alveolar border and anterior part of palate were repaired at the first operation and the lip and posterior part of the palate at later and separate operations. As our experience grows we are gradually adopting this procedure as the one of choice, reserving the first alternative to those cases where we wish to gratify the desire of the parents to have the lip closed as soon as possible. The "fourth alternative" is rarely employed in unilateral clefts of the alveolar border. It is reserved almost exclusively for bilateral clefts of Type II.

TABLE OF ALTERNATIVES

Operation.	First alternative.	Second alternative.	Third alternative.	Fourth alternative
First . .	Lip and alveolar border	Lip, alveolar border and anterior part of palate	Alveolar border and anterior part of palate	Alveolar border.
Second .	Palate, complete at one sitting (rare) or anterior part only	Palate, posterior part	Lip	Lip.
Third .	Palate, posterior part	. . . . .	Palate, posterior part	Palate, complete at one sitting (rare) or anterior part only.
Fourth .	. . .	. . . . .	. . . . .	Palate, posterior part.

We shall now proceed to describe step by step the operations included under the heading of the "first alternative."

First Alternative

First Operation. Repair of Alveolar Border and Lip. The first step is to separate the lips and cheek from the

underlying maxillæ over the greater part of their outer surface. Bleeding will be slight if the soft parts are pushed from the bones by sponge pressure after the mucous membrane is divided. Then the maxillæ are pushed together by thumb pressure

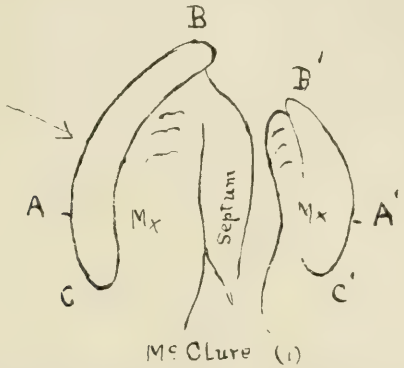

FIG. 6.—Life-sized tracing of a plaster mold of a case of complete unilateral harelip, cleft of the alveolar border and complete cleft of the palate. The lettering is identical with that used in Fig. 1. The arrow represents the direction of pressure in molding the maxillæ.

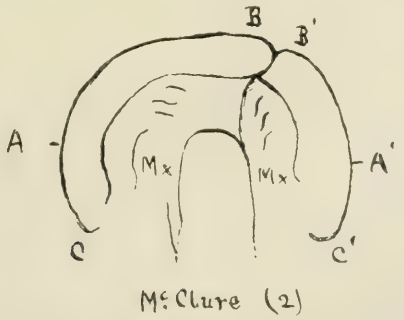

FIG. 7.—Same case as Fig. 6. Tracing of a mold taken some months after repair of the lip, alveolar border and anterior part of the palate.

exerted on the prominent maxillary process. Referring to Fig. 6, pressure is applied to the body of the maxilla in the direction of the arrow and the bone is pushed over until the distance AA' is reduced to its proper corrected length. The line of pressure

is changed, passing forward gradually toward the point B, until the ends of the alveolar margins, B and B', are brought into contact. Care must be taken to mold the body of the jaw, and not the alveolar processes, otherwise they may bend inward and the body of the jaw may remain in its original position. The molding must be done thoroughly, and we must not desist until most of the resiliency of the jaw has been overcome and the points B and B' show little tendency to spring apart. In children over three months of age digital molding alone may be impossible, and it may be necessary, first, to divide the body of the jaw by a transverse cut either by a knife or by a chisel.

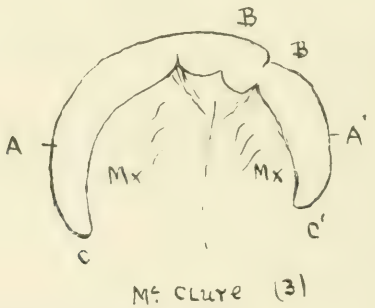

FIG. 8.—Same case as Fig. 6. Tracing of a mold of complete palate. The alveolar borders stand out in relief and the line of palatal union is well shown. The uvula made no impression on the wax mold.

The line of incision in the bone should be just below the malar buttress of the maxilla and it must always be above the sockets of the teeth. Afterward the parts are molded and brought together. Molding of the smaller side of the maxilla is seldom necessary, because its lateral displacement is slight. The coapted maxillæ are now fastened together. For several years I have been using kangaroo tendon to hold the bones in place. The mucous membrane is removed from the ends of the alveolar borders at B and B'. In doing this thoroughly we sometimes open the socket of a tooth, which necessitates its removal. A drill is passed through the body of each maxilla entering on its

outer surface above the sockets of the teeth and well behind the pyriform aperture of the nose. It enters the nasal cavity above the horizontal palatal plate. Along the track of the drill an Emmet needle is passed, carrying a silk thread which is used in turn to pull a strong strand of kangaroo tendon along the track. The maxillæ are finally brought close together and the tendon knotted in front. If care is taken, perfect approximation is secured. At this stage of the operation it will be found that

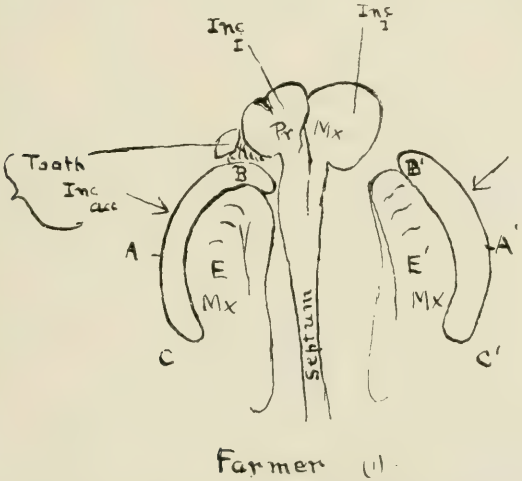

FIG. 9.—Life-sized tracing of a plaster mold of a case of double complete harelip, double cleft of the alveolar border and complete cleft of the palate. All the features are described in the text. The arrows show the direction of pressure in molding the maxillæ in the first operation.

the nose is straightened, the pyriform aperture of the nose restored and the sides of the alveolar border approximated. The margins of the cleft in the lip now lie quite close to one another. If the child's condition is good it is our custom to repair the lip at once. The edges of the cleft are pared and the operation is completed according to the technic described previously.

The results following the use of kangaroo tendon have been very satisfactory. It has failed me in one case only, and the

reasons for this were probably twofold, viz.: (1) Inadequate molding of the maxillæ; (2) the use of a strand of tendon too thin to stand the strain. It is absolutely necessary to mold until the resilience of the bone is completely overcome, otherwise the

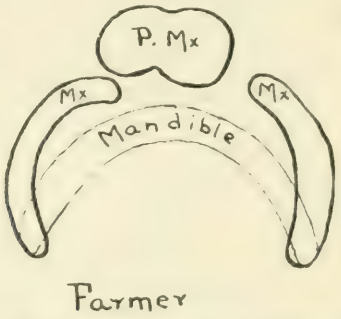

FIG. 10.—Outline sketch of superimposed alveolar borders of maxillæ, premaxillæ and mandible of Fig. 9, to show their articulation.

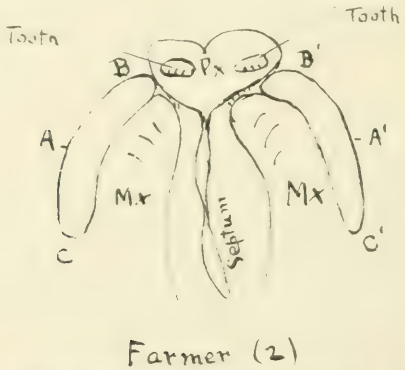

FIG. 11.—Same case as Fig. 9, showing a sketch of a mold of the palate taken after the jaws had been molded. This case was taken six months after the operation on the alveolar border.

kangaroo tendon will not stand the strain. If resilience cannot be destroyed it is far safer to fasten the bones together with silver wire, either with or without the use of lead plates. As a matter of fact the only reason for using kangaroo tendon is that it enables us to close the lip at the same sitting without the

worry of contemplating the subsequent removal of a silver wire from underneath the freshly united lip. In all two stage operations on the alveolar border and lip silver wire is preferable and safer.

Second Operation—Repair of the Palate. We allow a period of from four to six months to elapse between the operation on the lip and alveolar border and that on the palate. At this time we find that the palate is fairly accessible and that the lip will stretch to any reasonable extent. In favorable cases the palate can be repaired in one sitting. In the great majority two separate operations are required.

Fig. 12.—Same case as Fig. 9, showing sketches of molds of the palate in the various stages of repair. On the left, the result of the operation for repair of the anterior part of the palate. On the right, the result of a subsequent operation for the repair of the posterior part of the palate is shown. The steps of these operations are depicted in detail in Fig. 23.

1. *Complete Repair in One Sitting.* A tension stitch is passed on each side through the whole thickness of the anterior part of the soft palate. These stitches are retained until both soft and hard palates are completely mobilized. They are used exclusively as tractors and their employment reduces the handling and injury of the palate to a minimum. The mucoperiosteum of the hard palate is now divided down to bone by an incision (Langenbeck's) close to and parallel with the alveolar border. The incision should be about 1 cm. in length and should not

endanger either the anterior or posterior palatine arteries. Through this incision the mucoperiosteal layer is separated by an elevator from the bony palatal plate over its whole area of attachment, from front to back and from the alveolar margin to the edge of the cleft. The separation from the edge of the cleft can usually be done by the elevator, aided by occasional snips from sharply curved scissors. On the side of the maxilla to which the septum is attached care must be taken to avoid separating the mucous membrane from the side of the septum unless it is the intention to use it as a flap. A lateral incision through the septal and palatal union from front to back can be

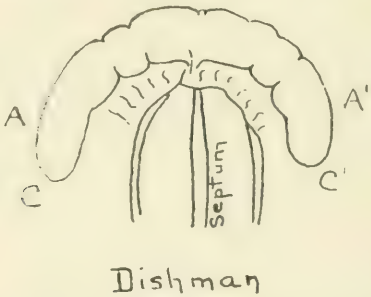

FIG. 13.—Life-sized tracing of a plaster mold of a case of complete cleft of the palate associated with intact alveolar margin and lip. Note the very wide cleft with vertical lateral palatal plates. The anterior end of the cleft is very wide and rounded. The septum hangs free.

made with a flat curved knife and this will facilitate the elevation of the palatal flap on this side. Next the attachment of soft palate to the posterior border of hard palate is divided. This (palatal aponeurosis) can be divided best by pulling on the tension stitch, elevating the soft palate and cutting the resisting tissue with curved scissors. At this stage both soft and hard palates can be lifted toward the operator and if the separation is adequate the flap will lie horizontal in the mouth. The same procedure is repeated on the opposite side. Too much emphasis cannot be laid on the importance of doing this part of the operation thoroughly. No important bloodvessel is imperilled if the

directions are followed as described above. At this stage, if the cleft is very narrow, the edges of the palatal flaps can often be brought together without tension, and after being pared they can be fastened together by a special technic (Brophy's plates)

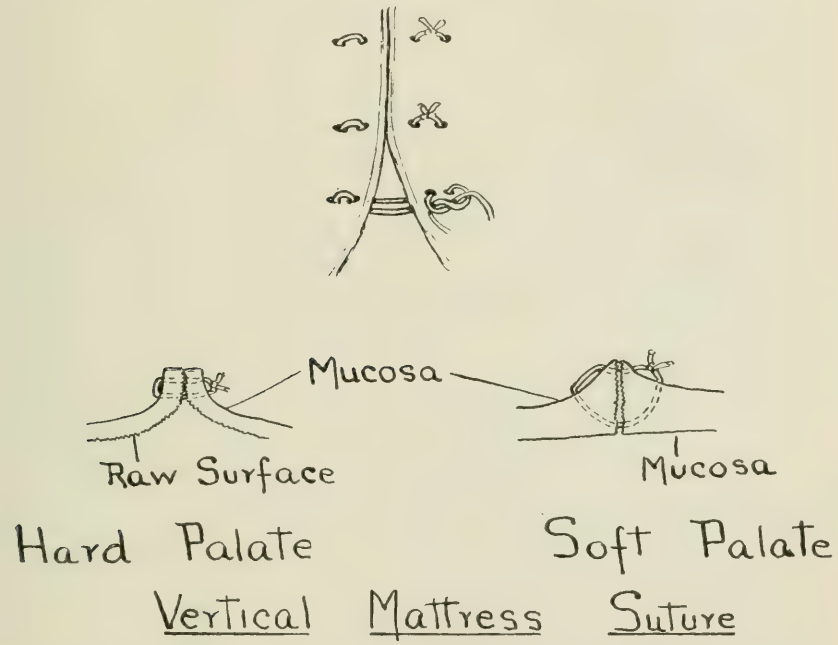

FIG. 14.—Shows the method of passing the vertical mattress sutures. In the upper figure the two upper stitches are tied very loosely; the lower one is not tied. It is clear that stitches passed in this manner cannot strangulate the blood supply to the edges of the flaps. In the lower figures, we see cross-sections of the stitches tied tight. On the left, the raw surfaces of the hard palate are seen to be held firmly together over a large area, and the edges turned out like the flanges of a water main. On the right, the stitch is shown passing through mucosa on buccal surface of palate, but not penetrating mucosa on cranial surface. This technic is of the greatest value.

with good prospects of primary union. (The essential features of Brophy's method are the following: Two silver-wire sutures are passed through the whole thickness of the soft palate on each side, penetrating it near its lateral attachment. The

ends of these wires are passed through holes punched in an oblong-shaped lead plate which is trimmed to fit the case. The plate rests on each side on the fleshy part of the velum internal to the hamular process. The edges of the cleft are pared and united by sutures from end to end. Finally the lead plates are adjusted and the silver wires tightened and knotted on each side. Great care must be taken to secure the right degree of tension on the plates. If this method can be carried out the functional results are much superior to those obtained by the methods to be described later, because the soft palate sustains no injury. Whenever possible, Brophy's method should be used. Unfortunately in very young babies conditions are rarely favorable and we are forced to secure complete relaxation by division of the soft tissues. We never hesitate, however, to use metal plates whenever there is any tension on the flaps.) In wider clefts the edges of the palatal flaps cannot be approximated at this stage without tension, and at any hazard this must be avoided. Tension can be lessened by increasing the length of the lateral incision. It can be carried forward a considerable distance without cutting the anterior palatine artery; and if care is taken to keep very close to the alveolar margin of the jaw and follow its posterior curve accurately it can be carried backward as far as the level of the hamular process without danger of wounding the descending palatine artery. It is our custom to carry the incision around the posterior margin of the alveolus in a short hook, and then to cut backward through mucous membrane only toward the middle of the tonsil along a line just lateral to the hamular process. The edges of the incision in the mucous membrane gape widely and the palate can be dislocated bodily from the maxilla toward the median line without cutting any of its muscles. If further liberation is necessary, division of some of the muscles of the soft palate is unavoidable. We believe that division of the muscles on the anterior surface of the palate is wrong in principle, because there is serious risk of wounding the radiating branches of the descending palatine artery if the buccal surface of the palate is incised.

The palatal muscles on the posterior or cranial surface of the velum can be divided without this risk. We have found that division of the palatopharyngeus and levator palati produces complete relaxation. They can be divided safely in the following manner. If the soft palate is elevated by the tension stitch, both palatopharyngeus and levator palati muscles can be brought

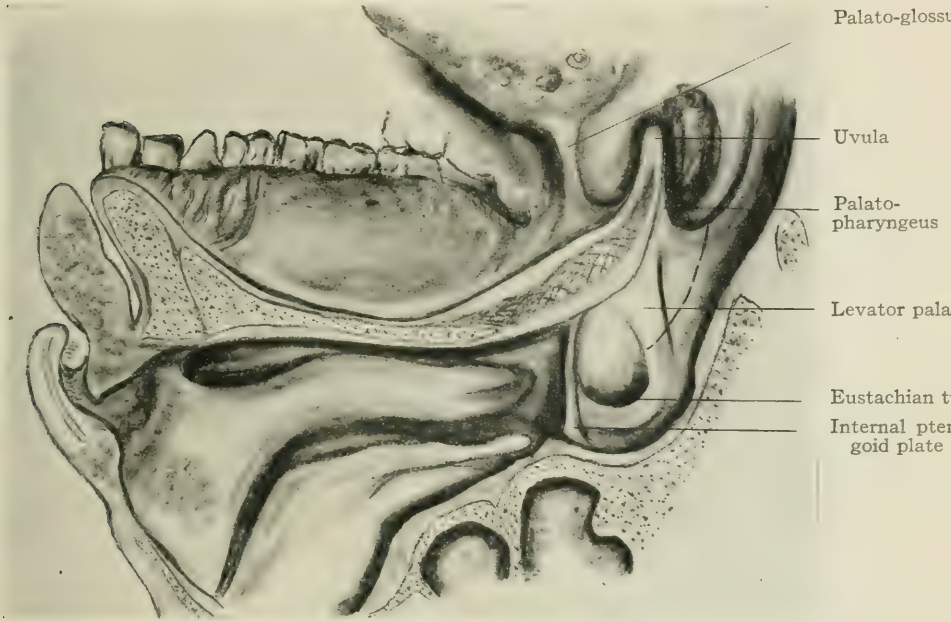

FIG. 15.—Original diagram of the soft palate showing the position of the palatopharyngeus and levator palati muscles. The dotted line indicates the position of the incision through the mucous membrane to relieve tension.

into view (Figs. 15 and 16). The palatopharyngeus muscle and the mucous membrane covering it are divided with scissors at a point just behind the middle of the tonsil. By cutting deeply into the lateral wall of the nasal passage with the scissors, along a line drawn from the divided palatopharyngeus to a point just buccal to the orifice of the Eustachian tube, the levator palati will be divided. Vigorous elevation of the velum by the tension

stitch will put this muscle on the stretch and it will produce a marked elevation under the mucous membrane.

Paring of the edges of the flaps is the next stage. In the hard palate this is unnecessary if mattress sutures are used; in the soft palate it must be done thoroughly and with the greatest care. The mucous membrane must be removed in a single, complete strip to avoid leaving small islands behind. On each

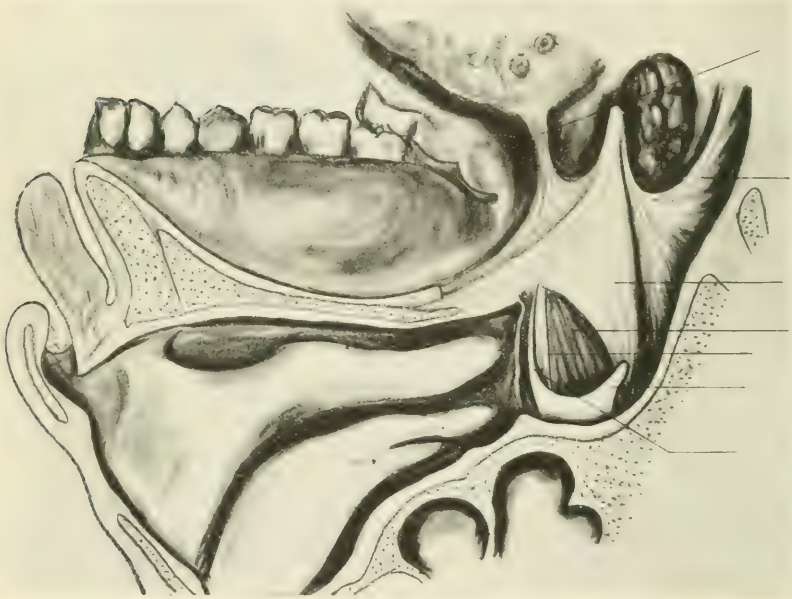

FIG. 16.—Dissection of the specimen from which Fig. 15 was drawn, showing the muscles. The labelling of the figure is explanatory.

side the soft palate is transfixed by a cataract knife and a thin strip of mucous membrane is removed from its free edge, extending from the tip of the uvula as far forward as the junction of soft and hard palate.

The last stage is the passing of the sutures. It is often convenient to secure control of each side of the palate by means of tension stitches. Usually we insert three on each side, one in

front, another at the junction of hard and soft palates, and a third through the thickness of soft palate lateral to the uvula. Blair prefers to handle the palate by means of a tenaculum made by fixing the broken point of a sewing needle in the jaws of a pair of forcipressure forceps. The first suture is passed through both edges of the cleft at the junction of hard and soft palates. This is not tied, but is merely used to lift up the flaps and to secure accurate spacing of the rest of the sutures. The sutures are now passed systematically from before backward. Each stitch is of the mattress type, inserted in such a manner as to avoid strangulating the blood supply to the edges of the flap (vertical mattress stitch, see Fig. 14). Three or four stitches may be required in the hard palate. When tied, the raw (upper or cranial) surfaces of the flaps are opposed to one another and the edges of the flaps are turned out like the flange in a water pipe. Similar stitches are passed in the soft palate, care being taken not to penetrate through mucous membrane on its cranial surface. By proceeding systematically from before backward, and using the last stitch passed as a tractor to hold up the flaps while the next stitch is inserted, little trouble is experienced in placing the sutures in good position. All the stitches are of the mattress variety except those in the uvula which are of the ordinary through-and-through variety. The suture material varies: For the mattress stitches we have lately been using carefully selected fine silkworm gut; for the other stitches we use horsehair or fine silk.

In Fig. 17 the steps of a palate operation, which was completed in one sitting, are shown. Reconstruction of lip, alveolar border and palate was finished at the age of six months. At the first operation, lip and alveolar border were repaired. At the second operation both hard and soft palates were repaired.

2. *Repair in Two Sittings.* Cases requiring the two-stage operation must be picked with the greatest care. They bristle with possibilities of failure, and if an unsuccessful operation is attended by sloughing of the flaps, subsequent reconstruction may be impossible.

It is a generally accepted fact that a cleft will become proportionally narrower and that the area of available flap will increase as time passes. Many cases with wide clefts and high palatal plates, which are quite inoperable in the first year of life, pass into the operable class at the end of the second year. Therefore it is wise to wait until measurements of the available tissue show that the operation can be undertaken with a reason-

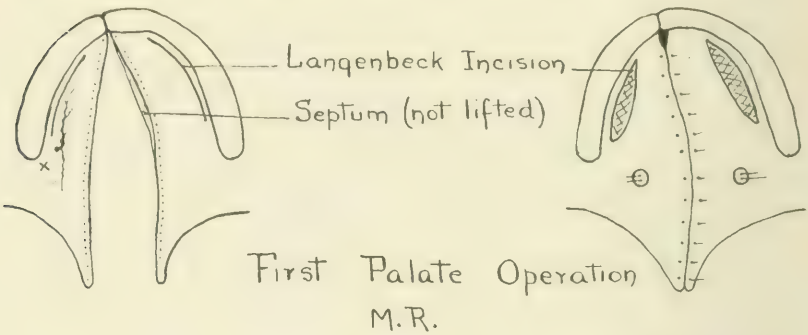

FIG. 17.—Sketch of the palate operation in case of M. R., reported in text, repaired from end to end in one operation. This case was reconstructed in two operations. In the first, the lip and alveolar border were repaired. In the second, the whole palate. Repair was completed at the age of six months. On the left, the position of the posterior palatine artery is shown in red coloring. The relation of Langenbeck's liberating side incision to the artery is clearly brought out. It is evident that the incision could be carried backward around the posterior end of the alveolar process without injuring the artery, if due care were taken to keep close to the alveolus. The cross is on the hamular process. On the right, the palate is shown with all the sutures in place. Small metal buttons with wire tension stitches are shown in the velum. In this diagram and most of the subsequent ones the velum is drawn unnecessarily long.

able prospect of success. In these cases it is better to divide the operation into two stages: first closing the hard palate and later the soft. It is impossible to lay down hard-and-fast rules of procedure, because each case must be judged on its own merits. It is better to attempt too little rather than too much, and to be content with closing the anterior part of the cleft without inflicting any injury on the velum; because, if the velum

is rendered hard and cicatricial, the last stage of the operation on the soft palate will be made very difficult and primary union problematical.

The steps of the *first stage* of the operation are essentially the same as those previously described. We always use the liberating side incision (Langenbeck) and lift the mucoperiosteal flaps

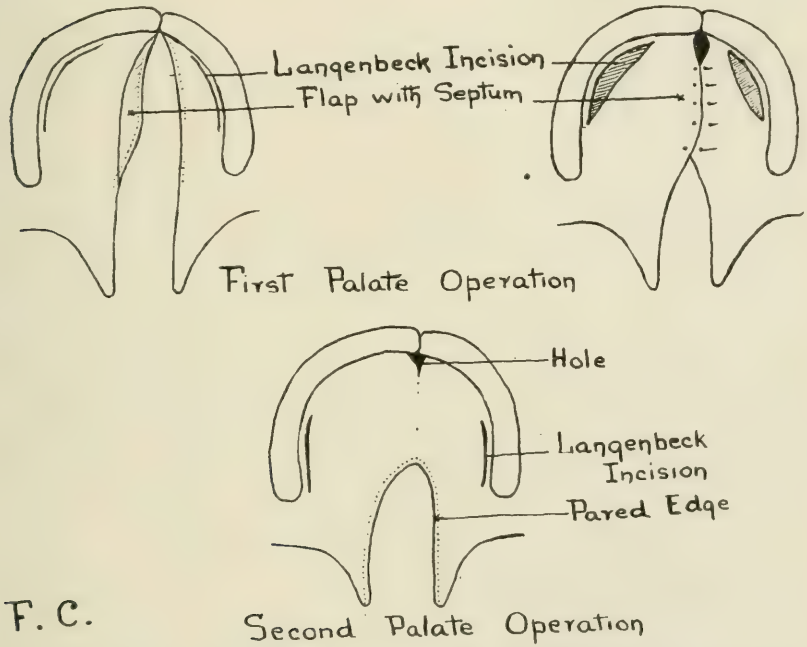

FIG. 18.—Palate operations in case F. C. The first palate operation left a hole in front, which is shown much reduced in size at the time of the second operation. An almost imperceptible hole still persists.

from the underlying bone very thoroughly. The palatal aponeurosis is divided and each flap is then freed. Of recent years we have made it a point to secure a flap from the side of the septum, as shown in Figs. 18 and 19. By this means the area of available flap has been increased materially. The edges of the two flaps are approximated and united by vertical mattress sutures as described previously. The sutures pass backward

only as far as the junction of hard and soft palate. The velum is not encroached on in the slightest degree. As a rule, union is sure and firm. The stitches very rarely cut out; at the worst, the hind stitch may give way.

The *second stage* is performed two or three months later. It is carried out in every detail as described previously. We find

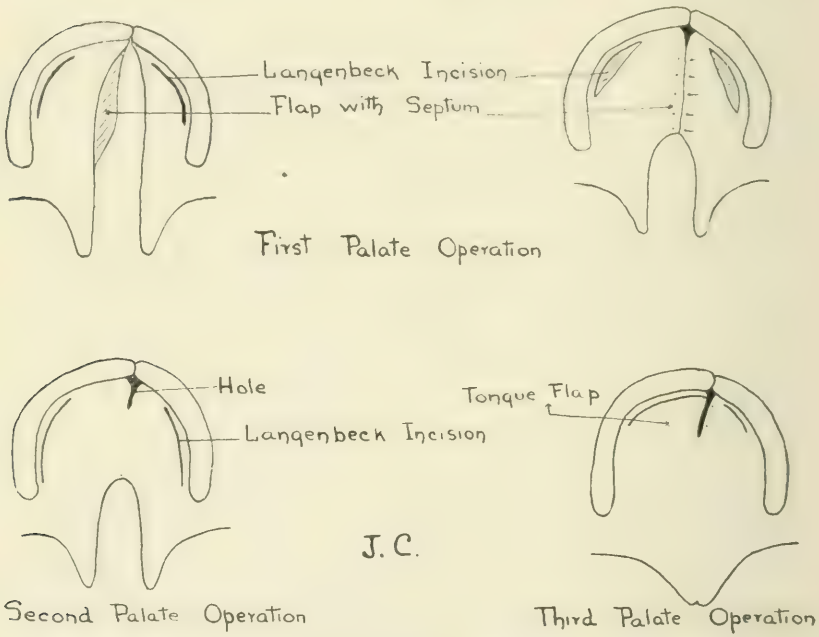

FIG. 19.—Palate operations in case J. C. The first operation left a hole in front. No attempt was made to repair this at the second operation, when the posterior part of the palate was repaired successfully. At the third operation the hole was closed by sliding a tongue flap across the gap from the right side of the palate.

it necessary to make large, liberating side incisions along the old tracks and through these the mucoperiosteum of the reformed hard palate is lifted up once again from its bony attachment and the palatal aponeurosis divided on each side. Not infrequently the side incisions are carried very far back and curved around the posterior end of the alveolar border. At any hazard

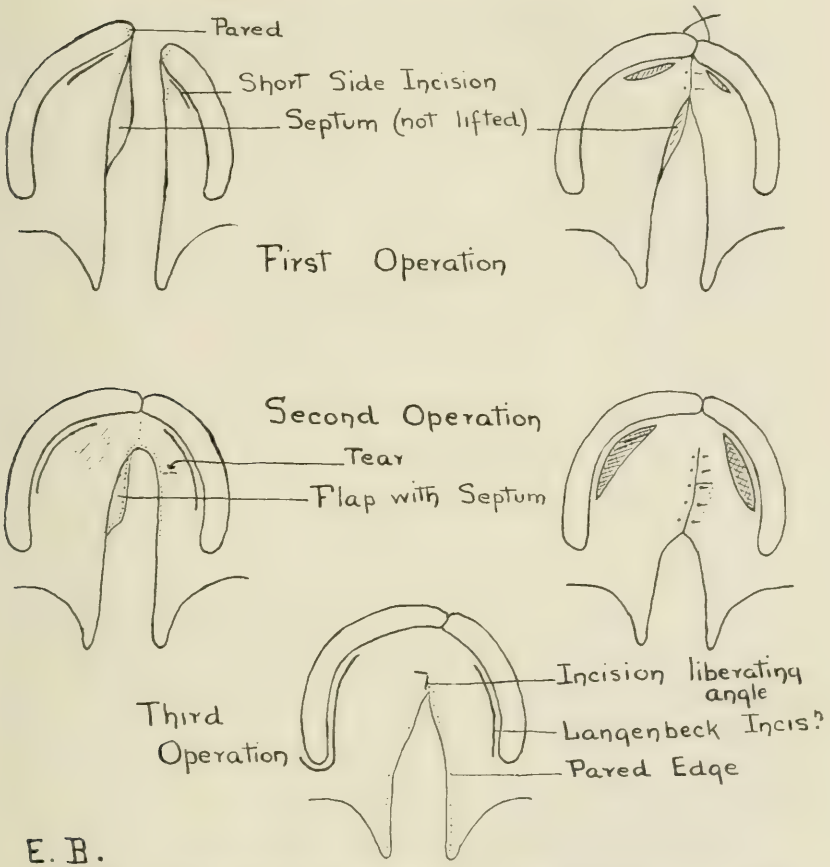

FIG. 20.—Palate operations in Case E. B. They show repair of the palate in three stages. At the original operation on the lip and alveolar border an attempt was made to close the anterior part of the hard palate by uniting the edges of the mucoperiosteal flap covering the palatal plates. Short Langenbeck's incisions were used. The septal flap was not used. The result was good. At the second operation the remainder of the hard palate only was repaired. A septal flap was used and very short Langenbeck incisions. The left flap sloughed and the result was a failure. At the third operation we employed very extensive Langenbeck's incisions; that on the right was carried in a curve around the end of the alveolar border. At the front end of the cleft we made an L-shaped cut to liberate the angle. The result was a success.

both hard and soft palates must be lifted from their beds until they hang free. Finally the levator palati and palatopharyngeus are divided and tension on the soft palate relieved, after which the edges of the cleft are denuded and the sutures inserted *secundum artem*. Figs. 18 and 19 with the legends attached are typical examples of cases operated on by the "first alternative," followed by two-stage operations on the palate. They are particularly instructive because each one was left with a small hole in the front of the palate just behind the alveolar border. This was closed in Fig. 19 by means of a tongue flap sliding operation.

Final closure of these small holes in front was so troublesome that we cast about for means to avoid the complication. In our first attempts the lip and alveolar border were repaired, and the anterior part of the cleft closed at the same sitting by elevating and sliding across the gap small mucoperiosteal flaps taken from the palatal plates. Such a case is shown in Fig. 20. Suture and adjustment of the flaps were very difficult and subsequent elevation of the mucoperiosteal layer in the later palate operation doubly so. Eventually we were led to use the mucous membrane of the septum in the flap and to formulate the sequence of operations comprised under the "second alternative."

Second Alternative

In this the first operation aims to mold the jaws, suture the alveolar border, repair the anterior part of the palate and close the lip in one sitting. At a subsequent sitting the remainder of the palate is repaired. The first case on which this procedure was tried was singularly successful. The first operation was followed by very little shock and the results were so satisfactory that reconstruction was completed at the age of five months. Fig. 21 shows the palatal procedures used in this case. In another case, shown in Fig. 22 the operation was successful apart from a slight failure of union at the junction of hard and soft palates, which necessitated a third operation for its repair.

The details of the method are as follows: Before the jaws

are molded, while the cleft is still wide open, a rectangular flap is reflected from the septum downward to the margin where the palatal and septal mucosa unite. To secure freedom of this

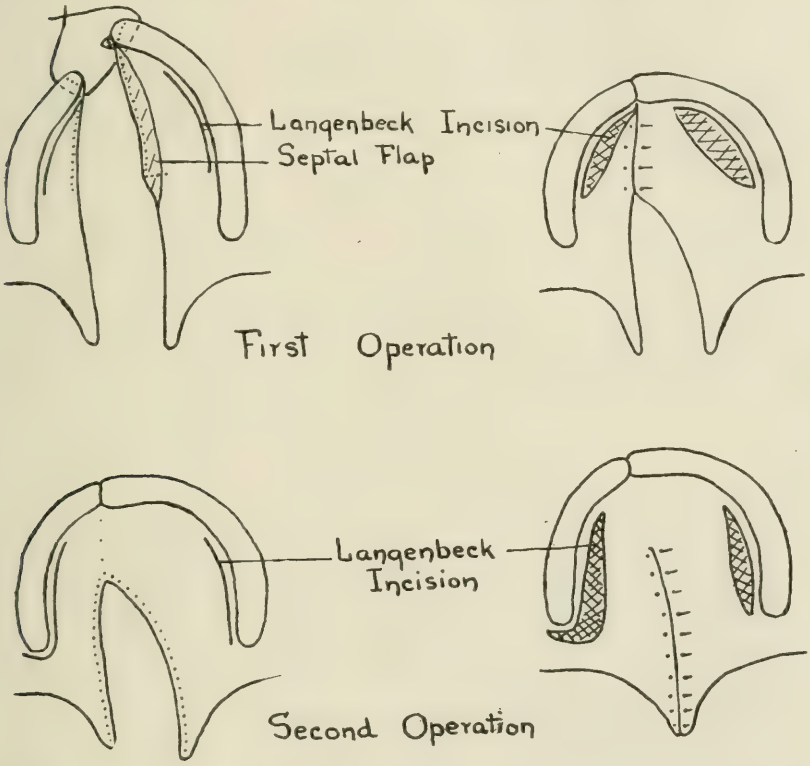

FIG. 21.—Palate operations in case J. H. In this case the anterior part of the palate was repaired successfully at the same sitting with the alveolar border and lip (second alternative). The posterior part of the palate was repaired successfully a few months later by using Langenbeck's incisions, as shown in the figure. Reconstruction was complete at the end of five months.

flap the mucoperiosteum is detached over a narrow area from the under surface of the contiguous palatal plate along the whole line of the septal attachment. This flap is carefully held up with tension stitches to avoid bruising. On the other side of the

cleft the edge of the mucoperiosteum of the hard palate is raised up along the whole length of its free margin and separated from

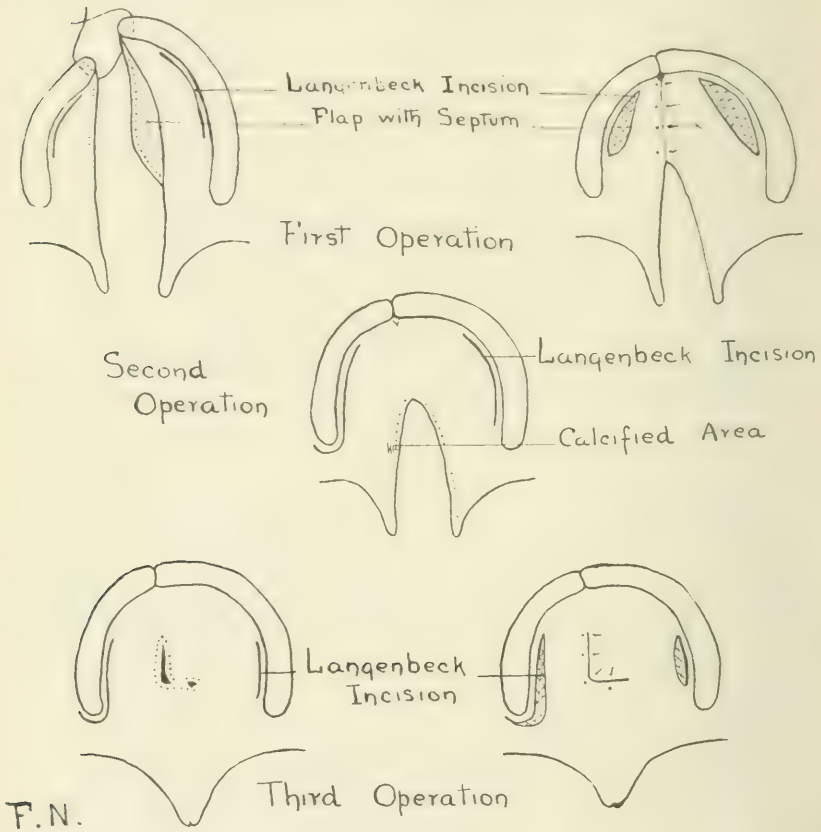

FIG. 22.—Palate operations in case F. N. At the first operation the lip, alveolar border and anterior part of the palate were repaired successfully. At the second operation the posterior part of the palate was closed successfully, with the exception of a part in the middle where two small holes were left. These were closed subsequently by a plastic procedure shown in the third operation. This necessitated extensive liberation of the palate by Langenbeck's side incisions.

the palatal plate until its deep raw surface is freely exposed. Langenbeck's incisions are used on each side. Tension stitches

are used to hold the edges of this flap. The jaws are now molded, approximated, and the kangaroo tendon suture passed but not tied. The edges of the alveolar margin are then pared. At this stage the raw surfaces of the palatal flaps are approximated and vertical mattress stitches of silkworm gut are inserted. If the flaps are long enough the stitches can be tied at once without danger. If, however, there is any tension, the tying of the stitches must be left until the last stage of the operation. Next the alveolar borders are approximated, the kangaroo tendon tied, and finally the palatal stitches are tied.

Our subsequent experiences with the procedure showed that the first operation is attended with serious shock. In one baby, death undoubtedly resulted from a miscalculation of the seriousness of the procedure. We, therefore, at the present time perform it only in babies who are exceptionally well nourished and robust.

Third Alternative

Gradually we are coming to the conclusion that the sequence of operations comprised in the "third alternative" offers the greatest degree of safety and enables us to secure the best cosmetic results. The only drawback is a sentimental one, in that we postpone the repair of the lip to the second operation and so prolong the suspense of the parents; otherwise, we divide the stages in an ideal manner and secure the most satisfactory reconstruction. At the first operation we repair the alveolar border, mold the jaws and repair the anterior part of the palate. The jaws are held together with silver wire (preferably) or kangaroo tendon. Brophy's side plates are used only in cases where molding is difficult and the resilience of the jaws cannot be overcome completely. The wires and plates are removed at the end of two weeks and the raw surfaces allowed to heal up completely before the second operation on the lip is performed. Four to six months after the lip has been repaired the posterior part of the palate is repaired *secundum artem.*

Fourth Alternative

Practically no cases of Type I in babies should be treated by sequence comprised under the "fourth alternative." A few neglected cases in adults would necessarily fall in this category. As stated before, the sequence becomes necessary in treating cases of Type II which comprise bilateral clefts of the alveolar margin and complete cleft of the palate.

TYPE II.—BILATERAL COMPLETE HARELIP; BILATERAL CLEFT OF THE ALVEOLAR BORDER; COMPLETE CLEFT OF THE PALATE

We have previously described the type of plastic operation that gives uniformly good results in cases of complete double harelip. Before this operation is undertaken it is necessary to correct the forward projection of the premaxilla and to place it in proper position between the anterior ends of the maxillæ. The outward displacement of the maxillary processes is first corrected. By thumb pressure aided by division of the bone by a chisel or knife, as may be necessary, the maxillæ are pushed toward the middle line until they are approximated to the right degree as verified by measurements with calipers. The premaxilla is then forced backward until it lies between and a little in front of their anterior horns. Removal of a wedge-shaped portion of the septum allows the bone to slip backward smoothly. Care must be taken not to jam the premaxilla too far back between the ends of the maxilla. It must always lie a little in front of the projecting edges of the latter bones. If placed too far back the profile view of the upper lip will be ugly and flattened. When these preliminaries are completed the mucus membrane is removed from the margins of the cleft on the front of the maxillæ and from the contiguous sides of the premaxilla. Finally the maxillæ and premaxilla are firmly united by strong silver-wire sutures fastened over lead plates

placed on the sides of the maxillæ, according to the method of Brophy. It is better not to attempt to close the cleft in the lip at this time because it is essential that this stage of the operation should succeed. In three or four weeks the silver-wires and lead plates can be removed, and as soon as the gums have healed completely and the mouth is surgically clean the lip can be repaired. At the second operation the lip is repaired *secundum artem*. We invariably use the method described earlier in this paper. The appearance of a double harelip immediately after the operation is never as satisfactory as that of a single lip. The nostrils are frequently unsightly, the openings of the nares look forward instead of downward, and the tip of the nose is often puckered downward by the short columella. As time passes these ugly features get smoothed out in a remarkable manner. A period of six months is allowed to pass before any operation on the palate is attempted. Careful measurements are made of the width of the cleft and of the amount of tissue available for flaps, and a judicial estimate made as to the possibilities of a successful plastic operation. If there is the slightest deficiency in the area of available flap the operation must be postponed without hesitation. However anxious we may be to close the palate before the child learns how to talk, it is far better to wait, rather than run the risk of failure. As time passes we find that the picture changes. The cleft gets narrower, the distance between the alveolar borders decreases and the area of mucoperiosteum on the palatal plates increases considerably. The following observations taken on one of our cases corroborate these statements in every particular (reference to Figs. 9, 10, 11 and 12 will explain the text).

The baby first came under observation in November, 1918. She was then five months old. At this time a mold was taken of both upper and lower jaws, life-sized tracings of which are shown in Figs. 9 and 10. (The letters A, B and C, and A', B' and C' refer to points on the alveolar borders that have been described previously. In speaking of the distances $A\,A'$, $B\,B'$, etc., we mean a transverse line drawn across the jaw between

the points A and A', etc.) The following measurements are recorded:

	Upper jaw.	Lower jaw.
Distance $A\ A'$	42 mm.	33 mm.
Distance $C\ C'$	42 "	39 "

On the lower jaw the points A and A' were difficult to fix accurately and therefore these measurements are only approximately correct. The difference between 42 mm. (for the upper jaw) and 33 mm. (for the lower jaw), which equals 9 mm. represented the amount of separation of the maxillæ. The average widths of the cleft were 13 mm. in front at $B\ B'$ and 16 mm. at the level $A\ A'$. On November 19 the maxillæ were molded and approximated and the premaxilla replaced. Brophy's technic was used. On December 12 the double harelip was repaired. On May 1, 1919, the baby was brought for inspection and a mold of the palate was made (Fig. 11). The following measurements were taken.

Distance $A\ A'$	36 mm.
Distance $C\ C'$	38 "

In addition further measurements were taken as follows:

1. Distance between the inner borders of the alveolar processes at the level $A\ A'$	30 mm.
2. Width of mucoperiosteum on the right palatal plate at the level $A\ A'$	12 "
3. Width of mucoperiosteum on the left palatal plate at the level $A\ A'$	13 "
4. Width of cleft at level $A\ A'$	15 "
5. Width of cleft at level $C\ C'$	12 "

From these measurements the following conclusions were drawn: (*a*) That the measurement $A\ A'$ had been reduced by 6 mm. and $C\ C'$ by 4 mm.; (*b*) that recognizing that the distance (1) 30 mm. must be bridged by flaps (2) and (3) which are respectively 12 and 13 mm. wide (= 25 mm.), we are still short 5 mm. of flap to bridge over the defect; (*c*) that the width of the cleft at $A\ A'$ was 15 mm., as against 16 mm. at the first measurement, showing a decrease of 1 mm.

Under the circumstances we thought it wise to postpone the operation on the palate until the child was older. On June 1, 1920 (a little over a year later) another mold was made. The following measurements were taken.

Distance *A A'*	35 mm.
Distance *C C'*	36 "
Width of cleft at *A A'*	8 "
Width of cleft at *C C'*	10 "
Width of right palatal plate at level *A A'*	13 "
Width of left palatal plate at level *A A'*	14 "

These figures showed that in a year's time the distance *A A'* had decreased 1 mm. distance, *C C'*, 2 mm.; in other words, that in spite of the general growth of the child's jaws there had been an actual decrease of the alveolar diameter of the upper jaw. They showed further an actual decrease in the width of the cleft of 7 mm. at the level *A A'* and of 2 mm. at *C C'*. Added to this we had a slight though appreciable increase in the width of the palatal plates by 1 mm. Allowing for the possibility of error in these measurements the evidence was clear that the jaws were nearer together, that the cleft was narrower and that the palatal plates were broader, and that the prospects of a successful operation had been improved by the delay. The anterior part of the palate was closed by a modified Langenbeck without difficulty.

As a rule, the palatal clefts are very difficult to close. I have never seen a case where the whole palate could be repaired at one sitting. As a rule it is safer to operate on the anterior end of the cleft and close it by a modification of Langenbeck's procedure, leaving the soft palate for a subsequent operation which should be postponed until the anterior part of the palate is completely consolidated and the flaps well vascularized. The method of closure will be easily understood by referring to Figs. 23 and 24. (Langenbeck.)

First stage: A liberating side incision (Langenbeck) is made in the hard palate on one side of the cleft. It is carried well forward and passes backward almost as far as the posterior

border of the hard palate. The mucoperiosteal layer is elevated thoroughly but the palatal aponeurosis is not divided. On the opposite palatal plate an incision is made which allows a tongue-

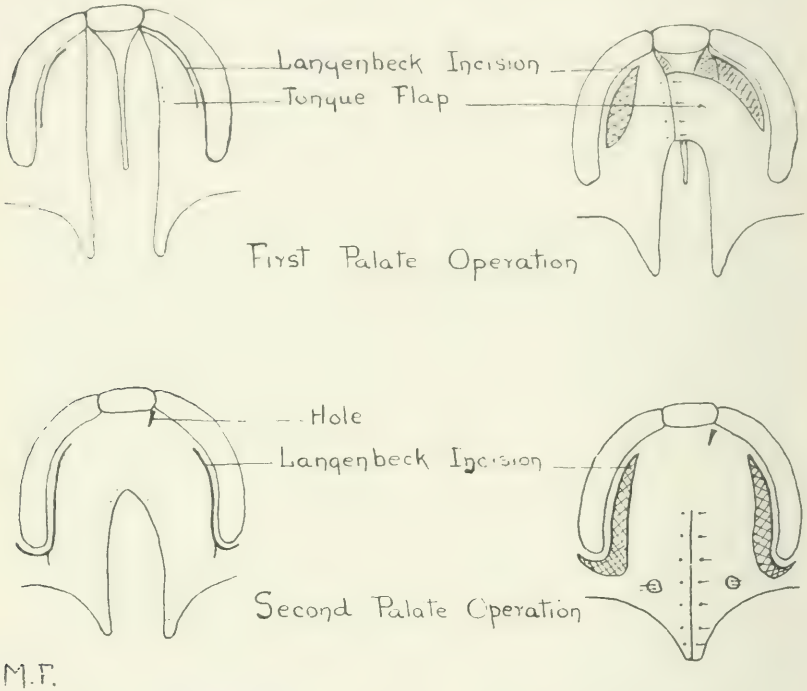

FIG. 23.—Palate operations in Case M. F. This was a case of double harelip and complete cleft palate. The anterior part of the palate was closed by a tongue-shaped flap which was lifted up from the left palatal plate, brought across the cleft and united with the edges of a flap lifted up by Langenbeck's method from the right palatal plate. The result was a complete success. A very small hole persisted in front. At the second palate operation extensive Langenbeck's incisions were used. Both were curved around the posterior ends of the alveoli and carried backward toward the base of the tongue. Brophy's tension stitches and metal buttons are shown in the soft palate. The result was a success.

shaped flap to be thrown up from the underlying bone.—The apex of this flap points forward; its base is attached behind. The tongue-flap is now slid across the cleft and sutured to the

flap lifted up from the palatal plate of the opposite side. As shown in the figure, a raw area is left in front, which fills up with granulation tissue. The result is usually very satisfactory.

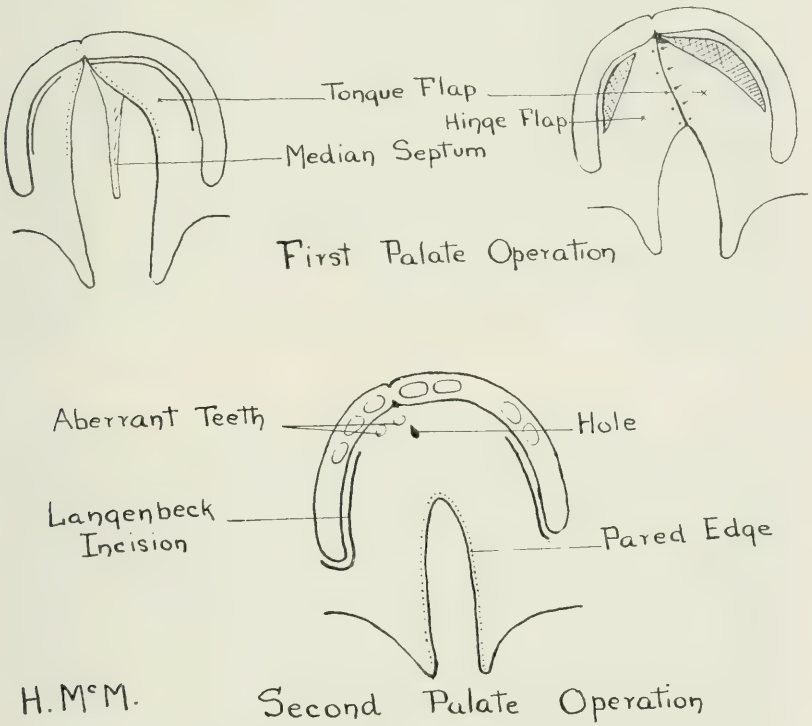

FIG. 24.—Palate operations in the case of H. McM. This was a case of double harelip with intact alveolar border. The palate and septum are shown in the upper figures of the first palate operation. The left maxillary palatal plate and septum had fused. The tongue flap was taken from the left side. The result was a success. At the second operation extensive Langenbeck's incisions were employed, that on the right curving around the posterior end of the alveolar border. Result, successful.

Frequently a small hole is left in front, which may give trouble (Fig. 23, first palate operation).

Second stage: Reference to Fig. 23 (lower left-hand corner) will show the steps of the operation. Liberating side incisions

are made. They are very free and encircle the posterior ends of the alveolar borders and if necessary are prolonged downward toward the root of the tongue. The hard palate is raised from the underlying bone with a periosteal elevator. It must be freed thoroughly, so that it hangs without tension and without being bound down to septum or palatal plates. During this part of the operation great care must be taken not to injure the descending [posterior] palatine artery. Then the soft palate is liberated by the method previously described, if necessary by division of the palatopharyngeus and levator palati. Finally the edges of the cleft are pared and sutured. Tension on the stitches may be relieved by the use of metal plates. It is our custom to place great confidence in them in these cases.

TYPE III.—LIP AND ALVEOLAR BORDER INTACT; COMPLETE CLEFT OF HARD AND SOFT PALATES

We have previously pointed out in this variety that the clefts are usually very wide and that the lateral palatal processes are high and vertical. The anterior end of the cleft is blunt and rounded. As a rule, the septum hangs free between the palatal plates. Very few cases are operable in the first year of life because of the extreme width of the cleft and the poverty of available flaps. As time passes the area of the palatal plates increases and the width of the cleft decreases. This favorable change continues slowly but surely until most cases gradually pass into the operable class. The type of operation best suited for these cases is the two-stage operation previously described under Type II. At the first stage the hard palate is repaired by sliding a tongue flap across the gap and uniting it to the edge of the opposite flap which has been raised by the Langenbeck method. The operation is confined entirely to the hard palate. A few months afterward, when the flaps forming the hard palate are thoroughly vascularized and consolidated, the second stage is undertaken and the remainder of the palate repaired *secundum artem*. Fig. 24 represents an example of this

FIG. 25

FIG. 26

FIGS. 25 and 26.—Case of double harelip and complete cleft palate showing result of lip operation described in text. The photograph of the repaired lip shows well-formed nostrils and a very satisfactory philtrum well supported by the premaxilla. The lower part of the lip is rather tight and drawn. This will be remedied when teeth appear in the premaxilla.

FIG. 27

FIG. 28

type, in which complete repair of a very wide cleft was obtained in two operations according to the method described.

The following abstracts of cases have been prepared to illustrate the nature and sequence of operations on the palate. They have been arranged to fit in with the scheme embodied in the table of sequences as first, second, third or fourth alternatives. We have purposely omitted reports of cases falling under the third alternative because the true value of this sequence is still *sub judice*. We are enthusiastic over the results obtained, but at the present time are not prepared to recommend it unreservedly.

First Alternative

Case I.—M. R., aged six weeks. Right complete harelip; cleft of the alveolar border; complete cleft of hard and soft palates.

First operation, July 17, 1918: Repair of lip; molding of maxillæ; repair of alveolar border; suture with kangaroo tendon. Result, perfect primary union.

Second operation (Fig. 17), December 11, 1918 (aged six months): Repair of whole length of palate; Langenbeck's side incisions; silver-wire tension stitches fastened over metal plates; mattress sutures of silver wire and horsehair. Result, primary union. A very small opening was left in front just behind the alveolar border. (December 27, the right nostril, which was too large, was narrowed by a plastic operation.)

Legend For Figs. 27 and 28.

Figs. 27 and 28.—E. B.—Case of single harelip and complete cleft palate. The photograph taken before operation shows the left flaring flattened nostril and marked deviation of the nose to the right. In the photograph taken ten days after the operation, at which time the jaws were molded and alveolar border repaired, the nostril is of natural size and the nose is straight. The red line of the lip has been restored and the lip is deep enough. The depression on the left of the line of union on the lip margin is not a notch but is produced by a slight twisting of the lip near the angle of the mouth.

Case II.—F. C., aged three months. Left complete harelip, cleft of the alveolar border; complete cleft of hard and soft palates.

First operation, December 10, 1918: Repair of the lip; molding of the maxillæ; repair of the alveolar border; suture of the maxillæ with kangaroo tendon. Result, primary union.

Second operation, June 28, 1919 (aged ten months): Hard palate repaired by taking a flap from the side of the septum; Langenbeck's side incisions used; mattress sutures of silver wire and silk. Result, primary union. A small hole persisted in front just behind the alveolar border. (See Fig. 18, first palate operation.)

Third operation, November 4, 1919: Repair of the posterior part of the palate. Small hole in the anterior part of the palate, almost imperceptible. Langenbeck's side incisions carried far back; mattress sutures of silver wire and silkworm gut. Result, primary union. (See Fig. 18, second palate operation.)

Case III.—J. C., aged one month. Right complete harelip; cleft of the alveolar border; complete cleft of hard and soft palate.

First operation, March 13, 1919: Repair of the lip; molding of the maxillæ; repair of the alveolar border; suture with kangaroo tendon. Result, primary union.

Second operation, June 6, 1919 (aged four months): Hard palate closed by using a flap taken from the septum; Langenbeck's side incisions employed. Result, good. Union complete along greater part of length of hard palate. A small hole persisted in front just behind the alveolar border. (Fig. 19, first palate operation.)

Third operation, October 23, 1919 (aged eight months): Repair of posterior part of hard and whole of soft palate. Langenbeck's side incisions. No attempt made to close hole in front just behind alveolar border. Result, complete healing. (Fig. 19, second palate operation.)

Fourth operation, February 25, 1920 (aged one year): Repair

of hole in anterior part of the hard palate. Langenbeck's side incisions and complete separation of flap A, which was made to slide over to the right. Result, complete healing. (Fig. 19, third palate operation.)

Second Alternative

Case I.—E. B., aged seven months. Left complete harelip; cleft of the alveolar border; complete cleft of hard and soft palate.

First operation, July 18, 1919: Repair of lip; molding of maxillæ after chiselling through malar buttresses; repair of alveolar border; suture of maxillæ with kangaroo tendon; repair of extreme anterior end of hard palate after liberation of flaps by small side incisions. Result, primary healing. (Fig. 20, first operation.)

Second operation, November 11, 1919 (aged ten months): Repair of whole length of hard palate by using septal flap and making Langenbeck's side incisions. Result, failure of union caused by sloughing of edge of the flap on the left side. (Fig. 20, second operation.)

Third operation, November 22, 1920 (postponed until baby was two years old): Repair of hard and soft palate at one sitting. Langenbeck's side incisions used. Result, excellent; primary union. (Fig. 20, third operation.)

Note.—In this case the attempt to close the anterior part of the palate at the first sitting was successful as far as it went. We succeeded, however, in closing only a very short length and did not use a septal flap. In the second operation the cleft was so wide that a septal flap was needed. The operation was very difficult. The result was that most of the line of union failed to heal. We never repeated the technic of the first operation. In subsequent operations we always used a septal flap. The case may be considered as a transition from the first to the second alternative.

CASE II.—J. H., aged one month. Right complete harelip; cleft of the alveolar border; complete cleft of hard and soft palate.

First operation, March 31, 1920: Repair of the lip; molding of the maxillæ; repair of anterior part of palate by a septal flap. Result, excellent; primary union. (Fig. 21, first operation.)

Second operation, July 28, 1920 (aged five months): Repair of posterior part of the palate; Langenbeck's side incision; double silver-wire tension stitches fastened over buttons; suture of silver wire and horsehair. Result, primary union; complete repair. (Fig. 21, second operation.)

CASE III.—F. N., aged six weeks. Right complete harelip; cleft of the alveolar border; complete cleft of hard and soft palates.

First operation, March 17, 1920: Repair of lip; molding of maxillæ; repair of alveolar border; suture of maxillæ by kangaroo tendon; closure of front part of cleft in hard palate by means of a septal flap. Result, primary healing. (Fig. 22, first operation.)

Second operation, July 10, 1920 (aged six months): Repair of posterior part of palate; cleft very wide; area of velum very narrow; Langenbeck's side incisions carried far back, especially on right side where it encircled posterior border of alveolus. Result, failure of union at junction of hard and soft palates; side of velum united. (Fig. 22, second operation.)

Third operation, November 4, 1920 (aged ten months): Condition as follows: Two holes present—one, anterior, showed as a median slit occupying posterior quarter of hard palate; the other, laterally placed at junction of hard and soft palates. (Fig. 22, third operation.) Palate closed after being liberated thoroughly by incision shown by dotted lines. The rectangular piece outlined by the dotted line was excised and the raw edges united by four silver-wire sutures.

Fourth Alternative

Case I.—M. F., aged five months. Complete bilateral harelip and complete cleft of the palate; vomer free; very prominent premaxilla. (For sketch of cast of palate, see Fig. 9.)

First operation, November 19, 1918: Molding of maxillæ and replacement of premaxilla after excision of a triangular piece from the septum. Silver wires and plates used according to Brophy's technic. Result, good.

Second operation, December 12, 1918: Repair of double harelip according to method described previously. Result, primary union; lip excellent. On May 1, 1919, a mold of the palate was taken, a sketch of which is shown in Fig. 11.

Third operation, June 1, 1920: The anterior part of the palate was closed by a modification of Langenbeck's operation. A tongue-shaped flap was lifted up from the left side and dissected up as far back as the junction of hard and soft palates. On the right side the flap was lifted up as in Langenbeck's procedure, the free edge being lifted up very carefully along the whole length of hard palate. After sliding the tongue flap across the cleft the edges were fastened together with mattress stitches. Result, primary union. (See Fig. 23, first palate operation.)

Fourth operation, April 14, 1921 (refer to Fig. 12, a sketch of a plaster mold): Repair of posterior part of palate. Langenbeck's liberating side-incisions carried backward and curved around the posterior end of the alveolar ridge; incision through mucous membrane carried from bend of curve toward tonsil; liberation of hard palate from bone, division of palatopharyngeus and levator palati muscles; silver-wire tension stitches fastened over metal plates; mattress sutures of silkworm gut. Result, primary union. (See Fig. 23, second palate operation, and Fig. 12, a sketch of a plaster mold of reconstructed palate.)

Case II.—R. H. Mc., aged eleven days. Double harelip; alveolar border grooved but not cleft; complete cleft of palate behind alveolar border; septum free; cleft unusually wide along

its whole length. Lip repaired at once (June 26, 1919). Result, good.

Second operation (on anterior part of palate), March 23, 1920: On right side of palate mucoperiosteal flap elevated through a long Langenbeck side-incision. On left side mucoperiosteal flap lifted up from bone, like a tongue, and separated from the posterior margin of bony palate. The tongue flap was carried sideways across the cleft and its free edge united by mattress sutures with that of the opposite side. No attempt was made to close the posterior end of the cleft. Result, primary union along the whole length of repair. (Fig. 24, first palate operation.)

Third operation, March, 1921: At this time a very small hole was present just behind the alveolar border; behind this the hard palate was firmly united to a point on a line of junction of hard and soft palates. Liberating side-incisions carried well backward around the posterior ends of the alveolar margins; division of palatopharyngeus and levator palati muscles. Cleft was very wide and palate had to be loosened very extensively. Suture with silkworm-gut mattress stitches. Result, primary union. (Fig. 24, second palate operation.)

DISCUSSION

Dr. J. J. Buchanan, Pittsburgh, Pa.:

We are very much indebted to Dr. Thompson for his paper. I cannot pass it by without strong commendation.

In this connection I wish to call attention to a device which I have used for many years, namely, a set of delicate knives and strong periosteal elevators in which the steel blades and handles have a flexible copper connection. This permits the angle of the blade with the handle to be altered at will and facilitates the lifting and paring of the edges of the flaps of the soft palate and uvula.

The alteration of the angle is easily and instantly made with the fingers.

One would think that the copper connection would weaken and break by many bendings, but I have had such instruments in use for many years (certainly twelve to fifteen). The blades have been ground many times but the copper is still intact. I commend these to you as practical instruments.

THE CAUSATION AND AVOIDANCE OF CEREBRAL DISTURBANCES IN LIGATION OF THE COMMON CAROTID ARTERY

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The danger to the brain in ligating the common carotid is well known and has been emphasized in all our text-books. For instance: "The surgeon is always in doubt whether the patient will come out of the anesthesia, and if he does whether he will exhibit hemiplegia or progressive cerebral softening" (Binnie), and again, "From the beginning of carotid surgery to the present time the dread of cerebral disturbances has invested the technically simple operation of ligating the common carotid artery with a gravity and anxiety that are associated with few other ligations" (Matas).

The percentage of cases in which the brain is affected has been placed as high as 54.5 (Le Fort), but 20 to 25 per cent is generally recognized.

The almost universally accepted cause is anemia with softening of the brain, from inadequate collateral circulation due to defects in the circle of Willis. This plausible theory is supported by (1) The occasional finding of such circulatory anomalies in fatal cases; (2) the possibility of producing cerebral symptoms by temporary compression of one carotid; (3) the more common occurrence of these disasters after middle life, which can be explained by lessened dilatability of the collateral vessels from increased rigidity.

The acceptance of the anemia theory naturally has led to various procedures to determine the presence or absence of an adequate

collateral circulation before resorting to permanent closure of the carotid, such as temporary ligation (Halstead, Jordan); gradual narrowing of the arterial lumen by encircling metal bands (Halstead, Matas), which is supposed to give time for the collaterals to expand; simultaneous ligation of the internal jugular (Ceci), with the idea of inhibiting cerebral anemia; and lastly, the abandonment of ligation in favor of aneurysmorraphy, or arterial suture, in the case of wounds, so as to preserve the circulation (Rehn).

Scant attention has been given to the objections to this time-honored theory. It fails to explain—

1. The interval which nearly always occurs between the operation and the advent of cerebral symptoms, which varies from a few hours to several days, or even two or three weeks. If anemia were the cause, symptoms should occur at once.

2. The suddenness of the onset of the symptoms. If produced by anemia, they should come gradually.

3. The greater frequency of brain complications in ligation of the common over that of the internal carotid. Theoretically, it should be the reverse, owing to the possibility of a retrograde flow of blood from the external carotid. This, in fact, has actually been observed to occur in certain operations in the neck.

4. Autopsies in which no arterial sclerosis and no deficiencies in the circle of Willis have been found (Zimmermann).

5. Cases in which the artery was temporarily compressed for a long time before operating, without ill effect, with disaster resulting after ligation (Goldhammer).

6. Cases in which a preliminary, gradually increasing constriction of the lumen by metal bands was resorted to, and still cerebral softening followed permanent tying of the vessel (Gruber and Werner).

7. A case (Perthes) where the common carotid was temporarily tied during the extirpation of a cervical tumor; but as soon as the ligature was divided and the circulation reëstablished hemiplegia followed, which cannot be explained by anemia.

Another explanation for softening of the brain following ligation of the common carotid was advanced by Zimmermann, in 1892,

and supported more recently by Stierlin, Perthes[1] and a few other observers. Although receiving scant recognition it has much in its favor and deserves serious consideration. It is based upon the fact that when the carotid is tied its inner coat is ruptured, which leads to the formation of a thrombus; and it is either the direct extension of this thrombus, or much more frequently the lodgment of detached emboli, which may block the cerebral arteries and cause anemia and softening. Rarely, when the intima is smoothly divided, thrombosis does not result; but where the edges are ragged or curl into the lumen of the vessel coagulation is inevitable.

The points in favor of this theory are numerous and convincing. It explains, for instance, why—

1. There nearly always is an interval between the operation and the cerebral disturbances, during which a clot is formed and emboli detached.

2. The brain symptoms come suddenly, like an apoplectic stroke, due to the lodgment of emboli.

3. Autopsies done in fatal cases following ligation, and also experiments upon animals, repeatedly have demonstrated the presence of ascending thrombosis and of embolism.

4. Tentative compression or temporary ligation, even when unaccompanied by symptoms, does not insure that disaster will not result when the artery is permanently tied, and neither does the gradual diminution of the arterial caliber by metal bands or otherwise.

5. Disaster is more frequent in ligating the common than the internal carotid, because the return flow from the external carotid is particularly liable to detach emboli from an extending thrombus in the main arterial trunk.

6. The release of a temporary ligature, with reëstablishment of the circulation, may initiate cerebral trouble, as demonstrated by Perthes.

The preponderance of softening of the brain in the aged is not difficult to understand because of the tendency to increased brittleness of the internal coat due to arteriosclerosis. This leads to increased likelihood of rupture from ligation and to roughness of the torn edges, thus increasing the tendency to thrombosis.

That ligation does not always produce trouble is clear when one remembers that the furrow produced in the inner coat by the ligature is sometimes so smooth that coagulation fails to occur, and that emboli do not always become detached even if a thrombus is present.

The fact that cerebral symptoms once in a while occur during an operation, so that the patient comes out of the anesthetic paralyzed, does not militate against the embolus theory. Such occurrences usually take place in connection with aneurysms, and admit of explanation on the ground that emboli have been dislodged by manipulation of the aneurysmal sac.[2] This has led Perthes to emphasize the necessity of closing the distal artery before the sac is touched.

Unquestionably it cannot be denied that anomalies in the circle of Willis occur; but it is significant that when they have been found after death following ligation of the carotid, it has not been proved that thrombosis or embolism were not present also. Nevertheless, in spite of theoretical considerations, we are not yet warranted in assuming that brain disturbances do not occur occasionally from anemia alone. Such an instance recently has been reported by Moser,[3] in which the marked symptoms resulting from a temporary ligation of the common carotid immediately were relieved by removal of the ligature. In such a case, however, a question must always arise as to the permanency and seriousness of the cerebral condition, and whether it may not have been due to interference with the sympathetic.

If this embolic theory is true, the older method of ligation should be discarded in favor of one which does not cause rupture of the internal coat, thus avoiding the formation of a thrombus. One way of accomplishing this is by the use of a metal band (Halstead, Matas), and another by the employment of a strip of fascia lata. The latter is preferable because it is not a foreign body, is less likely to injure the artery, and is always at hand when wanted.

The technic described by Perthes is simple (see illustration). A strip of fascia lata, three or four inches long and half an inch wide, is obtained from the thigh, wrapped once or twice around

the artery, tied in a half-knot, and the knot secured by a few sutures. It is pulled just tight enough to obliterate the vascular lumen without rupturing the internal coat. If desired, additional pressure and security against injury may be had by folding lengthwise another fascial strip several times upon itself and placing it between the knot and the artery. As is well known, fascia lata will not stretch or break easily, hence it makes an ideal material for the purpose.

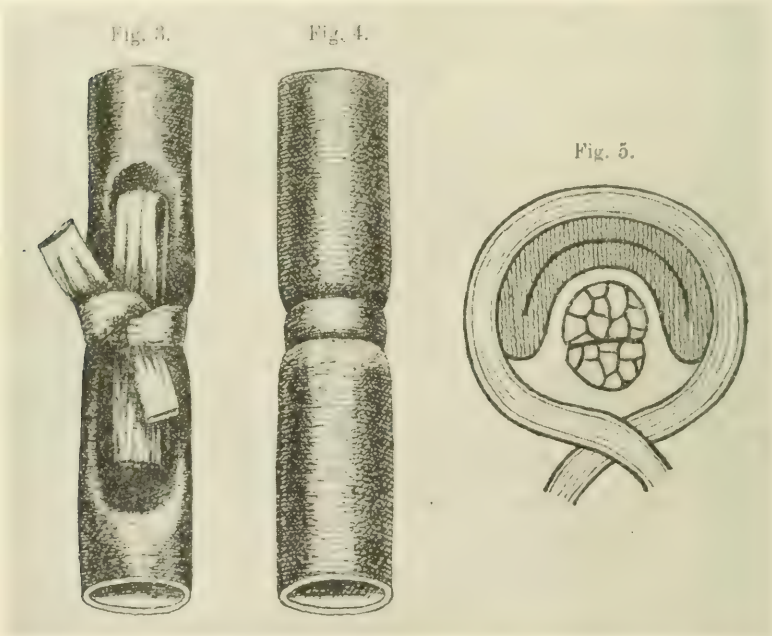

FIG. 1.—Ligation of common carotid artery with a strip of fascia lata, according to Perthes. (From Archiv. Klin. Chir.)

It goes without saying that aneurysmorrhaphy, as well as the suture of wounded vessels, cannot be discarded; but they should be employed only when they can be done easily and safely.

The following case is reported in order to add to the statistical material which will assist in deciding which of the two theories is the correct one:

A woman, aged fifty years, with a blood-pressure of 200, noticed, four or five months perviously, a beginning swelling just above the right sternoclavicular junction. This was accompanied by a disagreeable throbbing in the neck and neuralgic pains over the right side of the head posteriorly and the entire right shoulder. There was also difficulty in swallowing and a sense of choking. An *x*-ray picture demonstrated, however, that the dysphagia was due to a cardiospasm (pressure on the pneumogastric?) and not to direct interference with the esophagus. These disturbances increased rapidly and finally became so annoying that she applied to her physician, Dr. Alice Guthrie, for relief. Examination revealed a pulsating, expansile tumor, about as large as a walnut, which was evidently an aneurysm of the first portion of the carotid or of the innominate.

An operation was done, on January 3, 1921, in which was found a marked fusiform dilatation of the innominate, the first part of the subclavian and the first part of the carotid. Although the condition did not seem bad enough to justify a ligation of the innominate or subclavian, it nevertheless demanded that something be done to relieve the symptoms and prevent an increase of the trouble. Hence the following procedure was decided upon: Two strips of fascia lata were obtained from the thigh, each about four inches long and one-half inch wide. One of these was wound twice around the dilated innominate and secured to itself by stitches. It was drawn tight enough to lessen the caliber of the vessel without obliterating it, the amount of tension being regulated by the distal pulsation of the artery. By this means it was hoped to reinforce the vessel and prevent its further enlargement, according to the suggestion of Halstead.

The other strip was given a double turn about the lower end of the carotid and tied in a half-knot, which was secured from slipping by sutures of chromic gut. This permanent ligature was carefully drawn just tight enough to stop the distal pulsation without danger of rupturing the internal coat.

Convalescence from the operation was without incident, there being no cerebral disturbance of any kind. The pulse in the right

wrist remained normal, while that in the right common carotid was absent and remained so for at least two weeks.

The patient was relieved of all her annoying symptoms, including the throbbing, the cardiospasm and the various neuralgias. At the end of a month she was still in excellent condition and extremely grateful for the relief obtained. The pulsating and expansile tumor was no longer to be felt at the root of the neck, but a comparatively feeble pulse had returned in the carotid, due either to a collateral circulation from the back-flow through the external carotid or to reëstablishment of the circulation through the point of ligation. If deemed necessary, the former occurrence could be prevented by simultaneous ligation of the external carotid, and the latter by applying an ordinary ligature to the common carotid proximal to the fascial band and dividing the artery between them (Perthes).

Summary. 1. The great danger from cerebral disturbances in ligating the common carotid artery is universally recognized.

2. The accepted theory in accounting for this danger is that of anemia, due to failure of collateral circulation arising from defects in the circle of Willis.

3. An apparently more rational theory, recently emphasized by Perthes, is that of thrombosis at the point of ligation, followed by embolism. This accounts for the suddenness of onset of the symptoms and the greater or less interval which precedes them. The preponderance of cases after middle life is explained by the greater brittleness of the inner coat of the artery producing ragged edges when divided by the ligature and thus inviting thrombosis.

4. If this latter theory can be substantiated by experience it will do away with much of the fear and hesitation of the surgeon when confronted by this comparatively simple operation.

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1. Arch. Klin. Chir., B. 114, p. 406, Sept., 1920—Literature.

2. In this connection it is of interest to note that in one instance a patient upon whom I had performed a hysterectomy for a uterine fibroid came out of the anesthetic with a hemiplegia from which she subsequently died.

3. H. Moser, Zentralblatt f. Chir., No. 9, 1921, p. 321.

LIGATION (PARTIAL OCCLUSION) OF THE ABDOMINAL AORTA FOR ANEURISM

REPORT OF A RECENT CASE, WITH A RÉSUMÉ OF PREVIOUS CASES

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JUST one hundred and four years ago, Astley Cooper did the first operation of ligation of the aorta and the operation has been performed about nineteen times since, but in no case can it be said to have been successful in man according to the cases reported up to the present time. Death has always followed in from a few hours to a few months from shock, hemorrhage, infection or something directly connected with the operation, except Hamann's case, which died from hemorrhage from a bedsore.

Experiments on the lower animals (dogs, sheep and goats), by Luigi Porta, in 1838-40, both by partial and complete occlusion of the aorta by ligature, proved that such operations were by no means necessarily fatal, and he published some beautiful drawings of the collateral circulation established in animals whose aortas had been ligated more than a year before.

In more recent years the work of Halsted along the line of partial and gradual occlusion of large arteries by means of metallic bands has been of great value in demonstrating a method of occlusion without causing ulceration of the walls of the vessel.

In five of the twenty cases mentioned in this article, including my own, there was no ulceration, and in none of the five was there complete occlusion of the lumen of the aorta, and these

were the only patients, excepting Keen's, who lived long enough to give any expectation of complete recovery from the operation. Tillaux's patient lived thirty-nine days, Halsted's two patients forty-one and forty-seven days respectively, Hamann's six months and two days, and my patient is still living and working, one year and four months after operation.

H. B., male, white; aged thirty-nine years, bricklayer, was referred to me by Dr. James A. Gannon.

History. Had typhoid fever at ten years of age, "blood poisoning" at nineteen, and soon after that a chancre. For three years he has been suffering with pain in the abdomen and back. Two years ago his appendix was removed, and as he continued to have pain and developed a swelling in the abdomen just above the navel he was again operated on November 5, 1919, when the aneurism was discovered and the operation was carried no further. As the patient gave a positive reaction to the Wassermann test, he was treated for a while for syphilis. This had no appreciable effect on the aneurism; therefore I decided to try ligation of the aorta according to Halsted's principle of incomplete occlusion of the lumen of the vessel, using a piece of tape instead of the metal bands. January 23, 1920, the abdomen was opened, the aneurism, about one and a half inch in diameter, seemed of the saccular type and sprang from the left side of the aorta behind the pancreas about opposite the origin of the superior mesenteric artery. It was decided to place the ligature below the aneurism according to the Brasdor method. The peritoneum was opened, the aorta exposed and a piece of cotton tape one-half inch wide was carried around the vessel about two inches above its bifurcation and just below the origin of the inferior mesenteric artery. Two turns of one end of the tape made the surgeon's or friction knot, which was drawn gradually tighter and tighter until pulsation was no longer perceptible in the iliacs and barely so in the aorta below the ligature, then the knot was completed, the ends of the tape cut off and the abdomen closed.

Nothing unusual was noticed afterwards, such as increased

blood pressure and pains in the lower extremities when the patient recovered from the anesthetic.

Next day his condition was satisfactory—no pain or paralysis of legs, and the color and temperature were good. Pulse distinctly felt in left foot but none in the right—both equally warm and comfortable. A slight blowing sound could be heard by the stethoscope over the left external iliac, but none over the right. Pulsation was faintly perceptible in the left iliac but absent in the right.

Recovery from the operation was normal, but patient was kept in bed most of the time for two months and potassium iodide was given. When he left the hospital, March 30, the pain in back and abdomen was much less, the murmur of the aneurism was not as loud but was softer and more musical in character.

Some time during the month of May, against advice, patient returned to his work as bricklayer.

June 18, 1920, he was examined. Says he feels better than he has for years—no pain of consequence and he eats, sleeps and works well. The aneurism seems about one-half its original size. A soft whining sound is heard directly over the aneurism and a rough sound over the aorta just below—both systolic. No sound over external iliacs. A feeble pulse is felt in the left iliac and none in the right.

August 29, 1920. Examined patient. Thinks he is entirely well, no pain, has gained twenty pounds in weight, and does eight to ten hours' work bricklaying daily. The tumor cannot be made out by its pulsation. The circulation in the lower extremities is the same as before described.

February 13, 1921, when the last examination was made, one year and twenty-one days after the operation, everything seemed satisfactory except the patient's habit of going on occasional alcoholic sprees. He was hard at work and looking well. No aneurismal pulsation could be felt, but a soft whining note ending in a rough note below could still be heard in the region of the aneurism and the aorta just below. A faint pulsation could be felt in the left iliac and dorsalis pedis, none in the right iliac or

vessels below, yet the two feet were normal in color, temperature and sensation.

When last heard from in May, 1921, one year and four months after operation, patient was in good condition, but just recovering from a spree.

Below is given a résumé of 19 previous cases of ligation of the aorta and 5 cases of suturing or plugging of aneurisms of that vessel.

LIGATION. 1. June 25, 1817, Astley Cooper ligated the abdominal aorta for a left iliofemoral aneurism in a man, aged thirty-eight years. Death in three days.

2. July 5, 1829, James ligated the abdominal aorta for aneurism of the left external iliac artery in a man, aged forty-four years. Death from shock three and one-half hours later.

3. January 26, 1834, Murray ligated the abdominal aorta for right iliofemoral aneurism in a man, aged thirty-three years. Death in twenty-three hours.

4. August 5, 1842, Monteiro ligated the abdominal aorta for right iliofemoral aneurism in a man, aged thirty years. Death from hemorrhage ten days later.

5. June 21, 1856, South ligated the abdominal aorta for aneurism of the right common and external iliac arteries in a man, aged twenty-eight years. Death forty-three hours later.

6. March 8, 1868, William Stokes ligated the abdominal aorta just above the bifurcation for right iliofemoral aneurism in a man, aged fifty years.—silver-wire ligature. Death from shock twelve and three-quarters hours later.

7. March 30, 1868, Hunter McGuire ligated the abdominal aorta for aneurism of the left external and both common iliacs and the aorta, in a man, aged thirty years—the left ureter was included in the ligature. Death in a few hours.

8. August 6, 1869, P. H. Watson ligated the abdominal aorta for iliac aneurism in a patient, sex and age not given. Death from gangrene sixty-five hours later.

9. August 19, 1870, Czerny ligated the abdominal aorta (thinking it was the iliac) for gunshot wound of the thigh in a soldier aged twenty-seven years. Death in twenty-six hours.

10. January 19, 1879, Czerny ligated the abdominal aorta between the two renals for tumor of the left kidney in a man, aged fifty years. Death in ten hours.

11. July 3, 1890, H. Milton ligated the abdominal aorta just below the renals for a ruptured aortic aneurism in a man aged forty-five years. Death from anemia and shock twenty-four hours later.

12. December 12, 1899, W. W. Keen ligated the abdominal aorta just below the diaphragm, for ruptured aortic aneurism in a man, aged fifty-two years. Patient lived forty-eight days. There was no suppression of urine and the bruit in the aneurism did not disappear. Death from hemorrhage from the ligature cutting through.

13. January 20, 1900, Tillaux ligated the abdominal aorta just above the bifurcation for ruptured left iliac aneurism in a man, aged fifty-six years. Death thirty-nine hours later. He grew gradually thinner until death. There was no ulceration at the point of ligation and the lumen of the aorta was not completely obliterated, permitting the passage of a grooved director.

14. May 1, 1901, R. T. Morris occluded the abdominal aorta just above the bifurcation with a soft rubber catheter and clamp forceps for aneurism proximal to the ligature, in a woman, aged twenty-four years. Death fifty-three hours later from infection caused by gangrene of bowel in contact with the forceps.

15. June 20, 1904, A. Guinard ligated the thoracic aorta about the level of the ninth dorsal vertebra for aneurism of the end of the arch. Death three days later from suppression of urine. Guinard remarks that ligation above the renal arteries is incompatible with life, evidently not knowing the history of Keen's case.

16. January 1, 1905, W. D. Scott ligated the abdominal aorta just above the bifurcation for gunshot wound of the aorta and intestines, using a soft rubber catheter and forceps, in a negro woman, aged twenty years. Death one hour later.

17. December 18, 1905, W. S. Halsted applied his aluminum band to the thoracic aorta about 7 cm. above the diaphragm, partially occluding the lumen, for aneurism of the upper abdominal aorta. Pain was much relieved. January 10, 1906, twenty-

three days later, a second band was applied to the abdominal aorta just below the inferior mesenteric artery distal to the aneurism, just occluding the femoral pulse. Death eighteen days after the second operation from rupture of the aneurism. No ulceration at sites of bands.

18. February 23, 1909, Halsted applied his band to the aorta between the renal and superior mesenteric branches for aneurism extending from the renal arteries to the bifurcation of the aorta in a physician, aged fifty-three years, producing partial occlusion. Great relief from pain. March 5, there were enlargement of the aneurism and signs of infection, and Finney wired the aneurism March 12. Death April 11, 1909, from infection from a psoas abscess.

19. September 11, 1917, Hamann ligated the abdominal aorta just above the bifurcation for a pulsating tumor in the pelvis for which the right internal iliac had been ineffectually ligated, in a man, aged fifty-one years. Death March 13, 1918, six months and two days later, from hemorrhage from a bedsore. At necropsy no aneurism was found and the aorta at the site of the ligature was found only partly occluded, having a lumen less than three-sixteenths of an inch in diameter.

Suturing and Plugging of the Sac. 1. April, 1906, R. Lozano reports an operation on the abdominal aorta by Matas's endo-aneurismorrhaphy for aneurism. Death eight hours later from shock and hemorrhage—the diseased artery walls would not hold sutures well.

2. In the summer of 1906, John C. Munro ligated the right phrenic artery and the celiac axis and sutured with catgut the stump of the sac of an aneurism of the aorta above and behind the celiac axis, in a man, aged thirty-nine years. Death that night from hemorrhage.

3. July 30, 1904, Tuffier exposed by dissection an aneurism of the arch of the aorta threatening to rupture through the skin. No pedicle to the sac was found and adhesions were extensive, so extirpation was abandoned and the thorax closed. Death five days later, suddenly—the operation not having hastened his death.

4. October 29, 1909, Tuffier operated on a man, aged forty-nine years, the aneurism showing as a pulsating tumor in the right upper sternocostal region over which the skin was thin and adherent. In exposing the aneurism by turning back flaps, it was ruptured, and, to stop the bleeding, the tip of the gloved left index finger was thrust into the opening. A tampon was then made by stuffing the finger of a glove, the finger was withdrawn and the stuffed glove finger substituted and sewed into place and the wound closed with drainage. The patient was up on the eleventh day and walking on the thirteenth. Next day, the fourteenth, the tampon was replaced by a new one of the same kind. Two days later death occurred suddenly, but not from hemorrhage or asphyxia.

5. Kümmel reports (*Deutsch. med. Wchnschr.*, April 2, 1914) the case of a man, aged forty-five years, with an aneurism of the thoracic aorta just above the diaphragm. The aneurism was exposed by resecting three ribs close to the spine and just above the diaphragm, the aorta was controlled above and below with fingers, the aneurism slit open, the clots turned out, and the walls of the sac united with two rows of continuous sutures down to the lumen of the artery. Death in a short time from shock.

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A NOTE ON THE TREATMENT OF SECONDARY HEMORRHAGE FROM THE BRANCHES OF THE CAROTID ARTERIES

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It was a teaching of Kocher that to tie the common carotid artery in a person past middle life was always dangerous, and, in the arteriosclerotic equivalent to a death warrant.

It would seem that some surgeons tie this vessel rather indiscriminately and for somewhat trivial reasons, claimimg to see no ill results. Their luck is better than mine.

I have ligated the common carotid in but three subjects who were over forty years of age. One was for a carotid body tumor that completely surrounded the bifurcation and had materially reduced the caliber of the internal division. This patient survived withont immediate symptoms of brain ischemia, but the later history is not known. In the other two cases, although partial compression of the artery preceded the ligation for a period of more than twenty-four hours, both died with marked cerebral symptoms apparently referable to ischemia. An autopsy could not be obtained in either case.

I have ligated the common vessel about six times in people under thirty years, with only transient cerebral symptoms in one case; but I am so convinced of the dangers of this ligation in elderly people that I allowed one old man to bleed to death while making puttering attempts to control a secondary hemorrhage from one of the larger branches of the external carotid rather than tie the common artery, which I knew perfectly well to be the only sure method of controlling the bleeding.

It is an old teaching in English surgery, rather generally accepted, that when a true secondary hemorrhage occurs in the neck from one of the primary divisions of the common carotid, or from one of the primary branches of the external carotid near the bifurcation, only ligation of the common artery will prevent fatal hemorrhage. Death may occur within a few minutes or the first bleeding may be slight and cease spontaneously or in response to some minor proceeding, but it will recur and continue to recur. The reasons for this are that the vessel walls are held patent by the induration of the tissue and that the width of the vessel is so great compared to its length that the clot has no opportunity to organize. The secondary hemorrhage here referred to is that due to infection which causes the ligature to cut through the vessel wall, or the clot to be dissolved or blown out before permanent repair is established.

In my own practice I have not seen a true secondary hemorrhage in the neck, corresponding to the above definition, in over eight years, or since first heeding the advice of Billroth to neither suture any wound in the floor of the month nor to suture without free drainage any wound that contained a ligated carotid or a ligated primary branch.

I have had, however, two cases that somewhat approximated true secondary hemorrhage, the successful outcome of which suggested a plan that I hope may prove efficient in dealing with true secondary hemorrhage in this locality in people who are not safe risks for a ligation of the common vessel.

The first of the two cases just referred to was a hemorrhage following the throwing off of a slough after a complete removal of the tongue, with a cautery, right at its attachment to the hyoid bone. In this case the bleeding ceased spontaneously, showing that the fluids in a foul pharynx would not necessarily prevent the formation of a permanent clot in the open stump of the lingual artery.

It has been my custom for some time, in dealing with carcinoma of the tongue or pharynx, to ligate the lingual and facial vessels as far from their origin as possible, usually about an inch,

and leave the ligated ends protruding into the pharynx with no protection whatsoever.

FIG. 1.—Showing the appearance of the space left after the removal of the tongue and submaxillary strictures, after the author's method of radical operation for carcinoma of the tongue. (Note) Attention is particularly invited to the facial arteries standing up in the pharyngo-buccal cavity. They receive no further attention. The wound is closed by turning down and suturing the submaxillary flap of skin and platysma myoides muscle which is here shown turned up over the chin.

NOTE.—"Operation for Advanced Carcinoma of the Tongue or Floor of the Mouth." Surgery, Gynecology and Obstetrics, February, 1920.

The second of the two cases referred to above is the only one of the cases treated by the method just described in which a

delayed bleeding occurred and this was not a true secondary hemorrhage as above defined. Three days after removal of the tongue, and following a coughing spell, there was a persistent arterial bleeding, the blood coming from within the right side of the pharynx. On opening the right half of the submaxillary wound it was found that the stump of the facial artery stood up in the pharynx very much as it was at the time the submaxillary wound was closed at operation, and that just proximal to the ligation there was a fair-sized uncontrolled branch, the continued circulation in which prevented the formation of a firm clot in the stump of the artery. Further, it was found that another very minute branch that arose close to the preceding had been partially torn from the indurated wall of the stump and there was a pulsating stream of blood the size of a pin. No attempt was made to ligate the bleeding point, but a ligature was placed on the uncontrolled branch referred to above, and, after a few minutes' pressure on the stump, all bledding ceased. The wound was left open for observation and next day the stump of the facial artery, which had been nearly an inch long, was shrunken entirely out of sight behind the digastric muscle, and it gave no further trouble.

My conclusions from personal observations are:

1. That secondary hemorrhage of the carotid artery and its branches is to be prevented by not suturing wounds that extend through the floor of the mouth and by packing or freely draining all wounds in the neck, above the level of the thyroid cartilage, that contain a ligated primary branch of the carotid artery.

2. That secondary hemorrhage from a ligated primary branch might possibly be controlled by previously having placed the ligature as far from the carotid as possible, and when bleeding actually occurs, (*a*) to free the stump from the surrounding indurated tissues, (*b*) to ligate any branches that are given off proximal to this ligature and, if necessary, to loosely re-ligate the stump itself as far from its origin as possible.

If it is found necessary to place the ligature on the bleeding stump so close to the external carotid as to preclude the forma-

tion of a proximal clot, then the external carotid itself should be exposed and a ligature placed on each branch and on the trunk at least an inch from the bifurcation, in the hope of establishing a permanent clot in the external carotid itself. In this latter fashion I have successfully avoided ligating the external carotid dangerously close to its origin in a wound I knew would become infected.

DISCUSSION ON THE PAPERS OF DRS. FREEMAN, VAUGHAN AND BLAIR

Dr. Howard Lilienthal, New York:

Just a word to call your attention to something very important. I refer to the work of Dr. Harold Neuhof, of New York, on the use of sodium citrate solution intravenously and intramuscularly in order to reduce the blood coagulation time. His original paper was recently published in the *Medical Record*, and other papers on the same subject will follow. He has found that although *in vitro* the sodium citrate causes inhibition of coagulation, when injected into the tissues intramuscularly or intravenously in the human body, unless enormous doses are given, the exact opposite occurs. Nature seems to try to correct the tendency which the salt has to inhibit coagulation, and causes the organism to pour out the substances which are necessary for the formation of rapid clot.

We have used this method now in more than 200 cases. There was one accident in a case of carcinoma of the stomach, a case in which it was given intravenously and with too great rapidity, and the patient died. The method had been incorrectly applied in this case, the infusion having been rapid instead of slow.

I do not now use it intravenously but intramuscularly. It appears to be absolutely without danger; from 5 to 8 grams were injected; usually 30 cc of a 20 per cent solution. It reduces the coagulation time of the blood from one-half to two-thirds or even more. I have used it in many cases, and I use it in Mt. Sinai Hospital whenever troublesome hemorrhage is indicated. Its effects continue for twenty-four hours and even for forty-eight hours. By that time clots are formed and one need not fear secondary hemorrhage. There is no tendency toward slow coagulation when the effects wear off.

Dr. Harvey Cushing, Boston, Mass.:

If I have correctly understood Dr. Lilienthal in his news regarding secondary hemorrhage I feel that a still more important matter is the dislodgement of thrombi. Is it not possible that in our effort to accelerate clot formation we are going to do the very thing which we wish to avoid? It is dangerous to meddle with Nature's method in the formation of clots.

It is interesting that we should be still discussing a matter which, from the beginning of the history of surgery, has continued to be the operator's chief source of concern.

Unless bloodvessels are treated properly and handled delicately with as great avoidance as possible of mass ligation, thrombi which become easily dislodged will form. But the lungs are the organs that chiefly suffer, not the brain. It is evident from the recent studies that have been made on this subject that most postoperative pneumonias are due not to the anesthetic or to inhalation products but to the dislodgement of thrombi from small vessels in wounds. This condition may come on, as in the case of the more rare cerebral lesions, days or hours after the operation.

It is the most important technical matter with which we have to deal in surgery, the proper method of treating bleeding vessels. I cannot believe, however, that we are going to aid the situation by efforts to alter the general characteristic of the blood. We should depend rather on suturing than on ligating bloodvessels, and this is an important matter, particularly in the closure of wounds, in which end ligation of divided vessels which have a strangulated bit of tissue included with them should be avoided as far as possible.

Dr. George G. Ross, Philadelphia:

I should like to report a case of secondary hemorrhage following the incision of a cervical abscess. The patient was a boy, aged two and a half years, son of a doctor. The abscess, the result of a preceding tonsillitis, was ten days old when he reported at the hospital. An incision was made into the skin and the abscess cavity was opened by thrusting through the wall a pair of blunt-pointed hemostatic forceps. Pus immediately escaped, followed by a gush of blood. After a hectic twenty minutes the hemorrhage was controlled. It was discovered that the internal jugular vein had a perforated ulcer in its lateral wall, approximately one-eighth of an inch in diameter. The jugular was tied at both ends and the wound lightly packed with iodoform gauze. The patient was transfused with 300 cc of blood taken from the mother. Five days later a secondary hemorrhage occurred and arose from the external carotid artery. The vessel was ligated at both ends and the boy was again transfused. A culture from the cavity at this time showed pure culture of hemolytic streptococcus. Five days later a second secondary hemorrhage occurred, again from the external carotid artery. This had occurred shortly after I had left the hospital, to which I was hastily summoned to return. When I arrived the nurse was sitting alongside of the bed with her thumb on the bleeding-point. The patient was hurried to the operating room and the carotid was again ligated both above and below and another transfusion given, the mother being the donor. A culture taken at this time showed a pure culture of the hemolytic streptococcus. For fifteen days the lad

progressed in the most satisfactory manner and we thought his recovery was assured. The wound was granulating and there had been no further hemorrhage. Two days after his return home he developed a vomiting spell, followed by convulsions, meningitis, paraplegia and death. Prior to his death a culture taken from the spinal fluid showed hemolytic streptococcus.

Dr. John A. Hartwell, New York:

I wish to mention a case in which it was necessary to remove a portion of the common carotid in order to remove a tumor. After the operation the man had right-sided hemiplegia and aphasia, from which he finally recovered, and at the end of a year was able to walk and to use his arm to a great extent, but there was still some paralysis and aphasia present. There was no possibility of the liberation of a thrombus, because of the complete stoppage of circulation, unless one broke loose from the distal end of the internal carotid. It occurred immediately on awakening from the anesthetic, and probably was caused by cerebral anemia.

Dr. Howard Lilienthal, New York:

I agree with Dr. Cushing in the main with what he has said, but would call attention to the fact that the surgeon has to depend on blood-clot, no matter how produced, in checking hemorrhage. In cases of valvular disease of the heart, with much roughening, it might not be suitable, but in any event clotting within the uninjured vessel has never been noted in Dr. Neuhof's experiments.

Dr. John H. Gibbon, Philadelphia:

We should not close this discussion without congratulating Dr. Vaughan on his result.

This Association has witnessed for years the progressive work that has been done in the ligation of vessels and operations for the cure of aneurysms. I note the work of Dr. Matas and Dr. Halsted particularly. The case reported by Dr. Vaughan stands out unique, and I think that the Association should feel proud that such a result has been obtained by one of its Fellows. I still hope that the time will come when we will be able to apply the Matas technic to this type of aneurysm. I tried it once—the case was one of aneurysm of the abdominal aorta—and after controlling the vessels with a catheter I emptied the sac and was surprised to find that the introduction of three mattress sutures across the proximal opening into the aorta controlled the circulation completely. The patient, however, succumbed before the completion of the operation.

Dr. Martin B. Tinker, Ithaca, N. Y.:

All of us realize the anxiety which Dr. Freeman has mentioned in connection with cutting off the cerebral circulation when ligating the common carotid. A point which has not been mentioned, and which I believe is of value, is that we can save at least half an inch of the artery, which will help to carry on the collateral circulation, by suture instead of by ligature.

In excising malignant tumors, particularly of the carotid gland, I have twice first exposed the common carotid, put on artery forceps padded with rubber, which greatly facilitates the dissection of the tumor without bleeding, and then after dividing the artery sutured it, saving as much as possible of the vessel. The saving of even half an inch of the vessel will have a very great influence.

Dr. A. P. C. Ashhurst, Philadelphia:

I want to ask Dr. Freeman whether if the circulation after ligation of the common carotid is from the brain down through the internal carotid into the external, and not *vice versa*, as recent investigations seem to indicate—whether, if this is so, it will not somewhat alter our conception of the cause of the cerebral complications following ligation of the common carotid ?

Dr. A. Primrose, Toronto, Canada:

Some twenty-five years ago I assisted my chief, Mr. I. H. Cameron, to tie the right common carotid artery for the relief of an arteriovenous aneurysm of the back of the orbit. There was marked proptosis and great pain. A considerable degree of relief was obtained from the operation. Some four or five months subsequently the later Dr. R. A. Reeve brought the patient back to Mr. Cameron because the pain and distress had recurred. Dr. Reeve suggested that we should tie the common carotid artery on the opposite side. The second operation was carried out by Mr. Cameron. In this case, therefore, the common carotid artery was tied on both sides of the neck. An interval of some four or five months elapsed between the ligations. The man was twenty-six years of age and he recovered from the operative procedures without any apparent disturbance as far as the circulation was concerned.

With reference to the material used for wrapping about the weakened wall of a bloodvessel I desire to call attention to the special value of using fascia for the purpose. The value of a fresh transplant of fascia lies in the fact that it lives and becomes incorporated with the surrounding tissues in which it is embedded. The work of my colleagues, Dr. W. E. Gallie and Dr. A. B. Le Mesurier, is of interest in this connection. They have recently made a communication on the sub-

ject before the Orthopedic Society at their meeting a few days ago in Boston. Gallie and Le Mesurier have shown the value of fascia used as "living sutures" in a variety of conditions, such as hernia, recurrent dislocations of the shoulder, etc. They have shown experimentally and by histologic examination that the sutures actually live and become incorporated with the tissues in which they are placed. These observations would lead one to conclude that a fresh transplant of strong fascia about an artery, where it is necessary to support a weakened wall, would be an ideal procedure and would have distinct advantages over methods in which material foreign to the tissues has been used.

Dr. Stanley Stillman, San Francisco, Calif.:

In regard to Dr. Freeman's paper, I will state briefly that I have had occasion to ligate both common carotid arteries within a few weeks of each other without disaster, and I assisted Dr. Levi C. Lane, one of the early Fellows of this Association, in an operation for cancer in which he ligated the common carotid on the left side and three days later ligated the one on the right side. The man made a good recovery from the operation, but died subsequently as the result of a recurrence of the growth. I may say the second ligation was done for secondary hemorrhage.

Dr. Leonard Freeman, Denver, Col. (in closing):

As I mentioned in my paper, most if not nearly all of the paralyses have appeared immediately after the patients have come out of the anesthetic. These usually were cases in which aneurysms were operated upon and may be explained by supposing that manipulation of the sac dislodged the clot. I have had one case, however, in which the patient came out of the anesthetic with hemiplegia following a simple hysterectomy for a uterine fibroid.

Dr. Hartwell's case is certainly significant, but before regarding it as conclusive one must thoroughly understand that there were no clots in the carotid artery or no cancerous growths which might have been dislodged in manipulating the artery before it was tied.

I do not agree with Dr. Ashhurst. I think that experimentation, and also observations in operations upon the neck, have conclusively shown that after ligation of the common carotid the circulation may be from the external carotid into the internal carotid.

Dr. George Tully Vaughan, Washington, D. C. (in closing):

I have very little to say in closing the discussion except to thank Dr. Gibbon for his kind remarks. I do not feel especially puffed up over what has been done. I am not sure that the patient is permanently cured. However, he has lived longer than anybody else after ligation of the aorta.

OPERATION FOR THE CURE OF ANEURISM*

By ARCHIBALD MacLAREN, M.D.
ST. PAUL, MINN.

In preparing for an operation on a case of traumatic popliteal aneurism recently, I reviewed the literature on this rare condition, and found that the procedures advocated by Watson Cheyne, in 1900, and also by Bull, in 1904, had all been abandoned for the new operation advised by Dr. Matas. The cause of this change was due to the fact that in the older procedures so many cases of gangrene occurred or the cases which escaped gangrene were followed by a relapse of the aneurism.

Several modern text-books give the impression that reconstructive aneurismorrhaphy (Matas) was the operation usually employed. Illustrations showing the sacciform aneurism, depicting the false aneurismal sac, and also the picture of the fusiform sac, with the gutter in the bottom to mark the original vessel wall, were both featured in every text-book.

In Matas' second presentation of this subject before this Association in 1905, he complains that "great confusion and misapprehension still exists in the minds of many surgeons who had obtained their information from abstracts and second-hand descriptions given in the journals and text-books." In his report Matas listed 24 cases, 5 of his own and 19 by other operators. Of the total number, 18 were obliterated cases and 4 sacciform aneurisms where the lumen of the parent artery was restored by the closure of the single orifice of communication: 3 cases of fusiform aneurism were operated upon for restoration of the artery wall by arterioplasty—only 1 remained cured

* Read by title.

when last heard from at the end of eighteen months; of the other 2, 1 relapsed and 1 was operated upon again for obliteration of the sac two weeks after the arterioplasty.

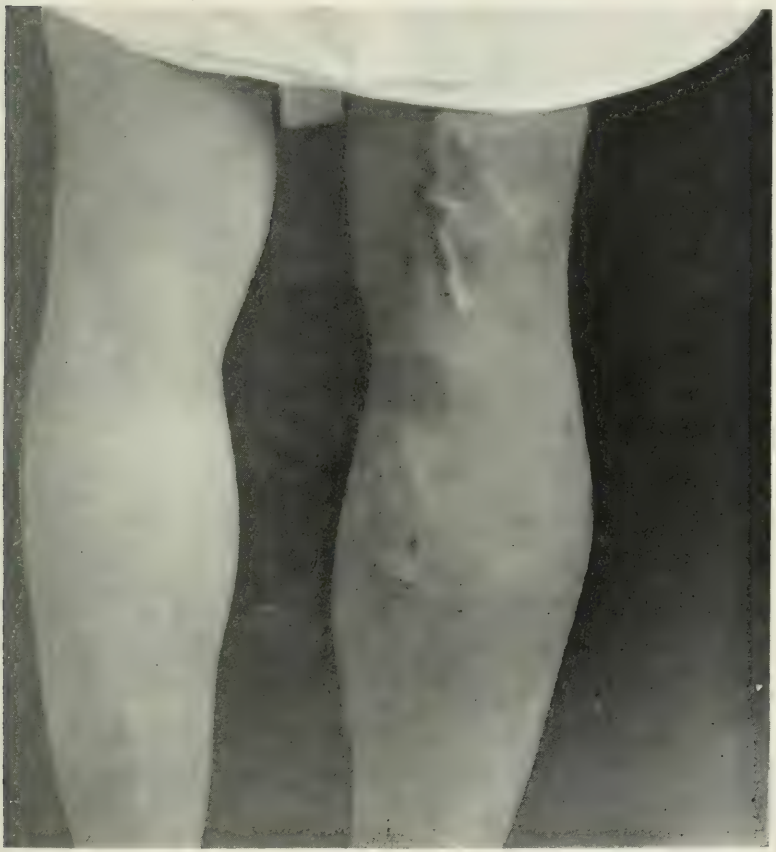


FIG. 1.—Showing swollen condition of affected leg.

Fortunately, for me and my patient, I came across a very sane and practical article on this subject by Dr. John H. Gibbon, of Philadelphia. Gibbon's conclusion, after a large experience with these operations, was, that arterioplasty or reconstruction of the vessel wall was seldom safe or necessary—that in practically all

cases the obliterative aneurismorrhaphy of Matas was the operation of choice.

E. H., aged twenty-two years; referred to me by Dr. Hopkins, of Arlington, South Dakota.

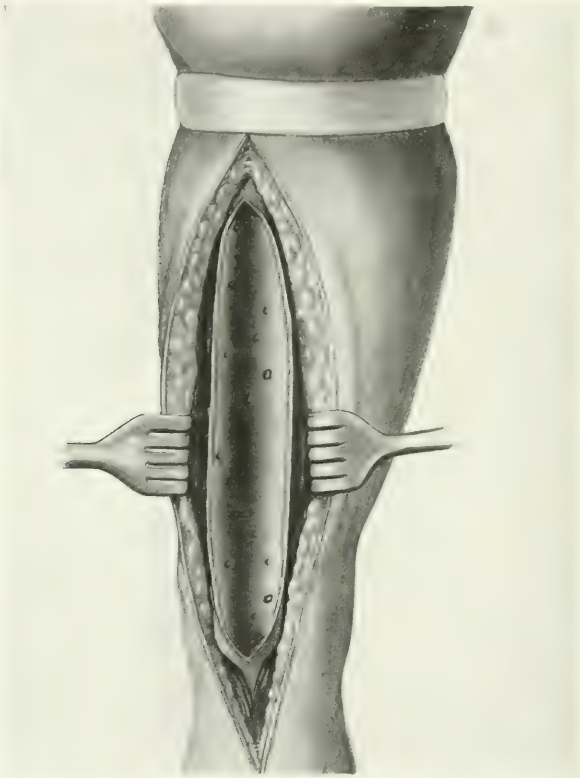


FIG. 2.—Fusiform traumatic aneurism of the femoral artery.

History. When six years of age, he had a large abscess in his left kidney region which was opened and drained. At the end of four weeks the drainage ceased and the opening was closed, and he had no further trouble with his kidney. When the boy was fourteen years old, which would be about eight years ago, he was accidentally shot with a .22-calibre rifle.

The muzzle of the rifle was about four feet away from the point of entrance. The ball struck him from behind in the upper part of the right popliteal space, probably injuring the arterial wall and imbedding itself in the internal condyle of the right femur. There was not much bleeding. His physician put him to bed for one week. He has not had any trouble with his leg until quite recently. He has noticed that the right leg was larger than the left, and that it tired more quickly. It might be interesting to note, that this boy entered the United States Army and served through the entire war, including service in France.

In the fall of 1920, he commenced to have a sharp pain in the right groin, especially when tired. He consulted Dr. Hopkins, who made an examination for hernia. No hernia was found, but there was a marked dilatation of all the superficial veins, especially in the foot and calf of the leg. There was a very marked thrill all through the leg, over every artery and vein. This thrill extended up into the iliac vessels. The upper leg was one and a half or two inches larger than the left. The most marked swelling and the maximum thrill were over the popliteal space.

Examination of abdomen and chest showed nothing pathological; urine and Wassermann were negative. The *x*-ray roentgenogram showed the bullet imbedded in the internal condyle of the right femur, but no necrosis or other diseased condition about the bullet.

The diagnosis of a large fusiform traumatic aneurism was made and operation was advised.

The operation was performed at the Miller Hospital, St. Paul, December 16, 1920. An Esmarch was applied about the upper thigh, just below the fold of the buttock. An incision over the dilated artery demonstrated a fusiform swelling of the femoral artery, extending up to the Esmarch and down to the lower end of the popliteal space. Upon opening the sac and cleaning out the clots we found many large vessel openings inside of the sac—about twenty in number. It was not possible to determine which one of these openings communicated with the vein; no gutter or other evidence of the original arterial wall was present; there

was no false sac. The entire thickness of the arterial wall constituted the aneurismal sac. Each one of the openings into the sac was separately sutured with fine chromic catgut.

The constrictor was loosened. One or two bleeding-points were reinforced with chromic catgut. Then the entire sac was obliterated by several continuous chromic catgut sutures. The collateral circulation was so good in this diseased leg that when the operation was finished the color and superficial heat were just as good, if anything even better, than in the normal leg. The thrill entirely disappeared and has never returned. He made a rapid recovery, was able to walk in three weeks, and is now well except for a slight keloid condition of the scar. Dr. Hopkins reported on March 16, 1921: "The leg is swollen below the knee, but above that point is normal." On June 2, Dr. Hopkins writes: "Regarding the present condition of our patient, I would say, that he is in good shape; the wound is entirely healed, and there is no evidence of a relapse of the aneurism."

A SURGICAL APPROACH TO THE SPHENOPALATINE* GANGLION

By CHARLES H. FRAZIER, M.D., Sc.D.
PHILADELPHIA

That there are certain pain phenomena of the face that are not attributable to lesions of the trigeminal nerve, no one will question. That some of these pain syndromes are relieved by cocainization of the sphenopalatine ganglion I have demonstrated on many occasions. The sphenopalatine ganglion derives its sensory supply from the maxillary division of the trigeminal nerve, but the conventional operations upon the trigeminal tract give absolutely no relief to these atypical neuralgias.

Since the sphenopalatine ganglion has intimate connection through the large superficial petrosal nerve with the sympathetic system, one wonders whether the pain of these neuralgias may be of sympathetic origin. There is nothing very speculative in this supposition, since in other territories there are painful conditions in the origin of which the sympathetic system is an acknowledged factor.

Attempts to relieve these neuralgias by alcoholic injection of the sphenopalatine ganglion, as proposed by Sluder, meet either with only partial success or with failure. Knowing the minute dimensions of the ganglion and the difficulty of access to it, one can readily understand why the results of injection are in many instances unsatisfactory and bear no analogy whatsoever to alcoholic injections of the divisions of the Gasserian ganglion. Having given much thought and time to the study of these neuralgias, presumably related to the sphenopalatine ganglion,

* Read by title.

I had rather reached the conclusion that unless a method for extirpation of the ganglion was at our command we had reached

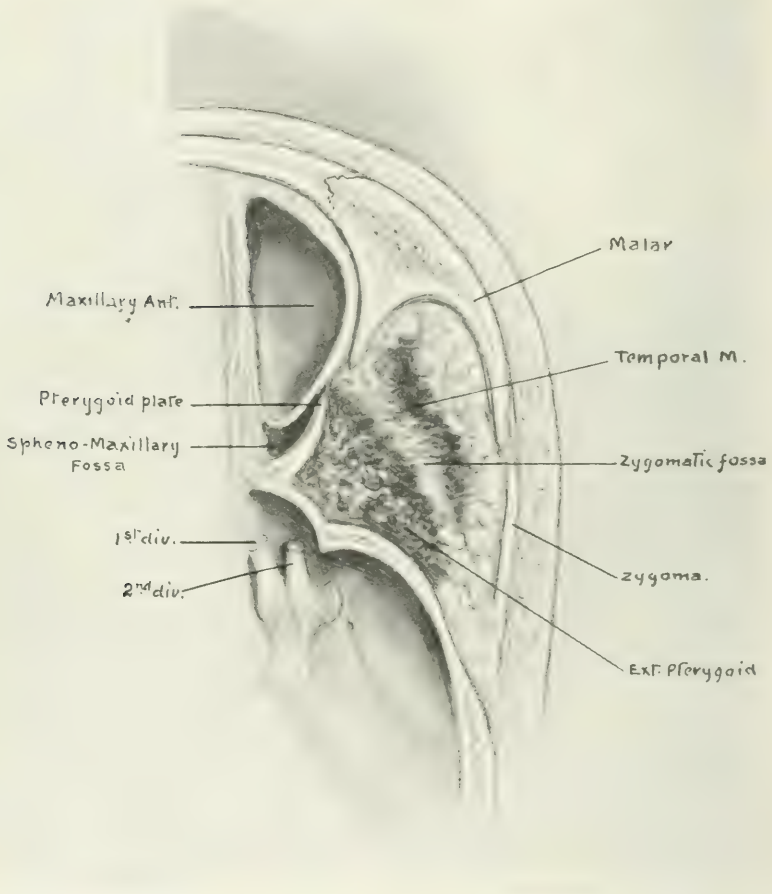


FIG. 1.—Horizontal section. Anatomical relationship of the structures of the zygomatic and sphenomaxillary fossæ.

an impasse. Only by the extirpation of the ganglion in a series of appropriate cases will we be able to speak with any positivity either as to the etiology or as to the treatment. If there is a

clinical entity—call it Meckel's neuralgia if you will—the extirpation of Meckel's ganglion should be curative in effect, should it not?

Upon this assumption, therefore, my assistant, Dr. Francis C. Grant, and I have been at work in an attempt to find a way of approach to the ganglion which would make it accessible for

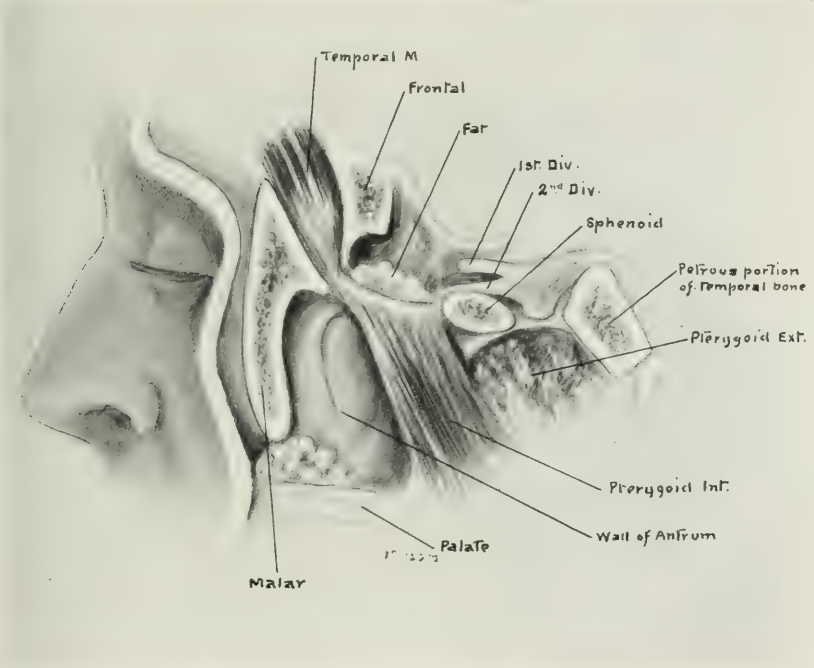


FIG. 2.—Vertical section. (*a*) Relationship of the structures in the zygomatic fossa.

purposes of extirpation. The approach we now present, I venture to hope, will prove a practical solution of this problem. In the two instances in which the operation was performed, there proved to be no inherent technical difficulties in gaining access to the fossa in which Meckel's ganglion is located. The anatomical landmarks (Figs. 1, 2 and 3) and the various steps in the

procedure as portrayed in the illustrations (Figs. 4, 5, 6, 7 and 8) require little amplification in the text.

In the first three illustrations (Figs. 1, 2 and 3) will be seen the anatomical structures and relationships. The most direct route has been chosen as the most favorable and practical

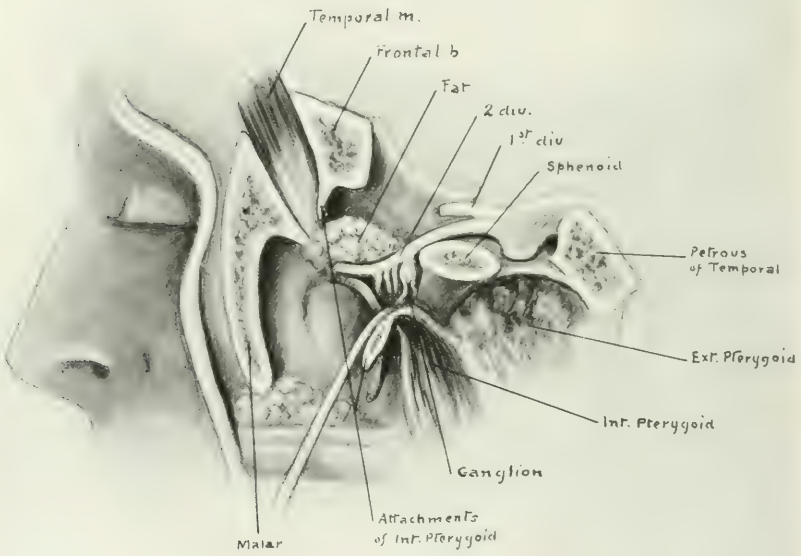


FIG. 3.—Anatomical relationship of the second division and the sphenopalatine ganglion in the sphenomaxillary fossa.

approach—a route that transverses first the zygomatic, then the sphenomaxillary fossa. To render accessible the zygomatic fossa the malar bone must be resected. Within the zygomatic fossa one encounters no obstacles not readily disposed of. All that one sees is a mass of areolar tissue, readily displaced, the posterior wall of the antrum, and the internal pterygoid muscle.

To reach the sphenomaxillary fossa, in which is lodged Meckel's ganglion, a portion of the external pterygoid plate must be removed. Once within the sphenomaxillary fossa there are no structures to be seen except the maxillary division of the trigeminus, the artery that accompanies it, a mass of areolar tissue which surrounds Meckel's ganglion and its several branches.

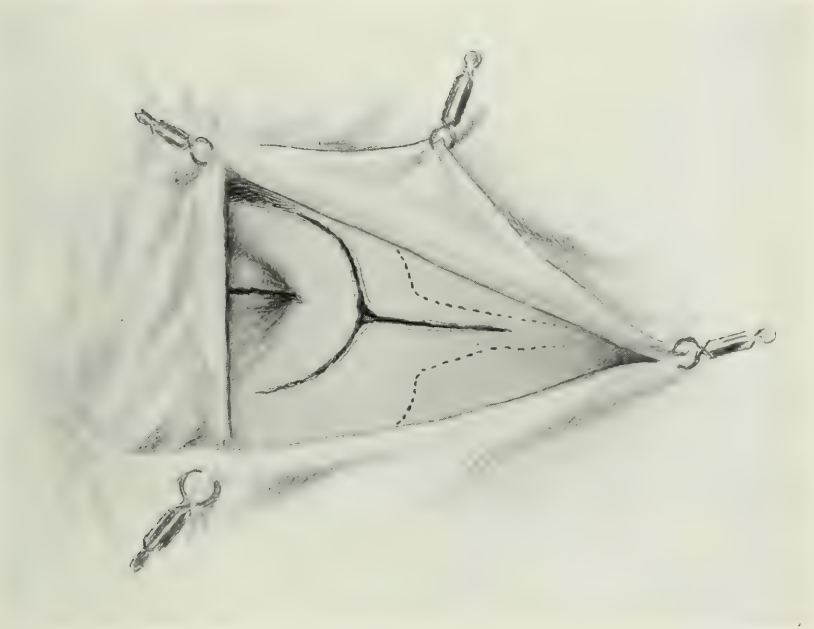


FIG. 4.—The incision in relation to the orbit and the zygoma.

STEPS OF THE OPERATION

I. The incision (Fig. 4) has been designed with due regard for its cosmetic effect and to avoid important branches of the facial nerve. There are three limbs, one straight, in the direction of the zygoma, and two curved, following the lines of the supra- and infraorbital ridges. With careful apposition of the margins of the wound the healed scar is quite inconspicuous.

(See photograph.) The branches to the orbicularis palpebrarum and the occipitofrontalis have not been disturbed.

II. Upon reflection of triangular flaps (Fig. 5) the malar bone is exposed and with a Gigli saw three sections of the bone are

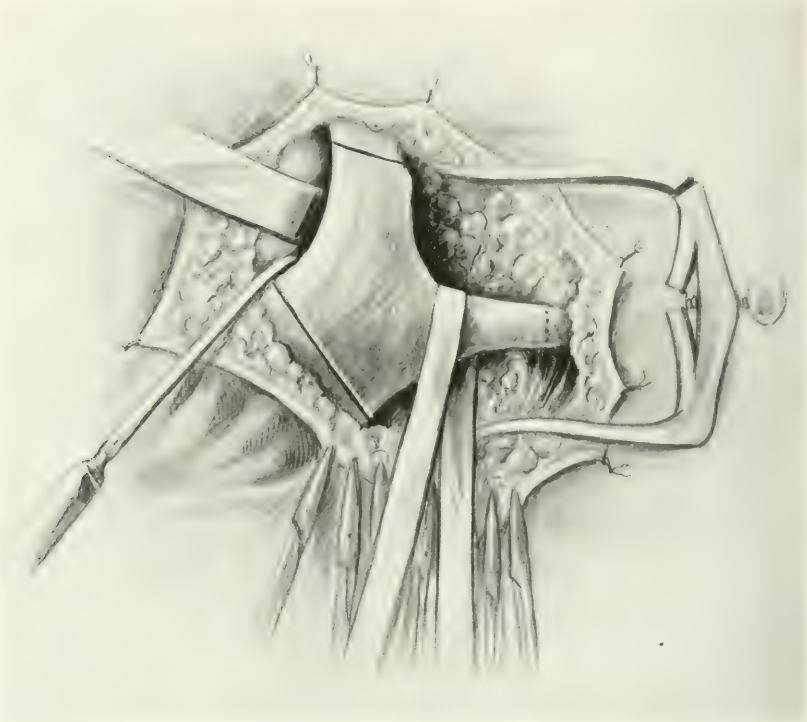


FIG. 5.—The isolation of the zygoma and malar bone. (*a*) Malar bone sectioned above and below. (*b*) Dotted line represents the point at which zygoma is divided with Gigli saw from within outward, leaving the outer periosteum intact.

made: (1) Through the frontal process; (2) through the maxillary process, and (3) through the zygomatic process. To make sections 1 and 2, the Gigli saw is passed through the sphenomaxillary fissure. At section 3 the zygomatic process is sawn only partly through, the outer shell and the periosteum

being left intact. Thus an attachment is conserved which prevents any dislodgment of the malar bone when replaced at the completion of the operation.

FIG. 6.—The malar bone reflected backwards uncovering (*a*) the fat and muscle tissue in the zygomatic fossa; (*b*) a portion of the orbital contents.

III. The malar bone reflected backward (Fig. 6) at once exposes to view the zygomatic fossa and its areolar tissues. One sees in the anterior portion of the wound the external aspect of the orbit.

IV. A clearing of the contents of the zygomatic fossa is made now to expose the pterygoid plate (Fig. 7). This is accomplished by following closely the surface of the posterior wall of the antrum and displacing backward and downward the areolar tissue and the temporal muscle. Before the pterygoid plate is exposed to view the internal pterygoid muscle must be detached.

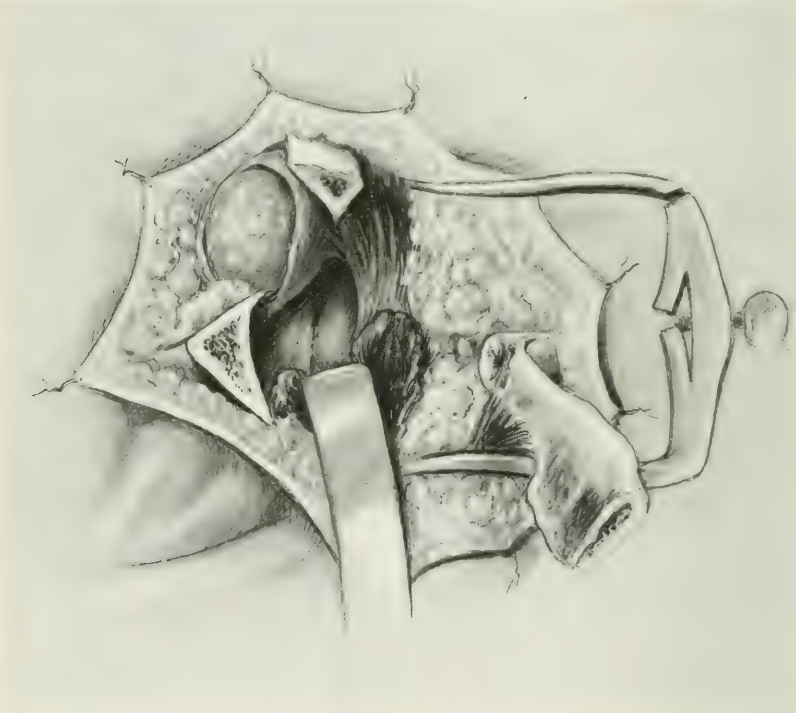


FIG. 7.—(*a*) Temporal muscle is retracted downwards exposing wall of antrum. (*b*) Internal pterygoid muscle above, intact.

V. With rongeur forceps a portion of the pterygoid plate is removed and the contents of the sphenomaxillary fossa exposed. To find the sphenopalatine ganglion one should expose first the maxillary division, as it enters the orbit through the spheno-

maxillary fissure, and follow it up to the ganglion. The ganglion itself is deeply placed in the sphenomaxillary fossa, close to the sphenopalatine foramen. Surrounded by fat, it is not readily seen; hence the necessity of following the course of the maxillary division as a guide.

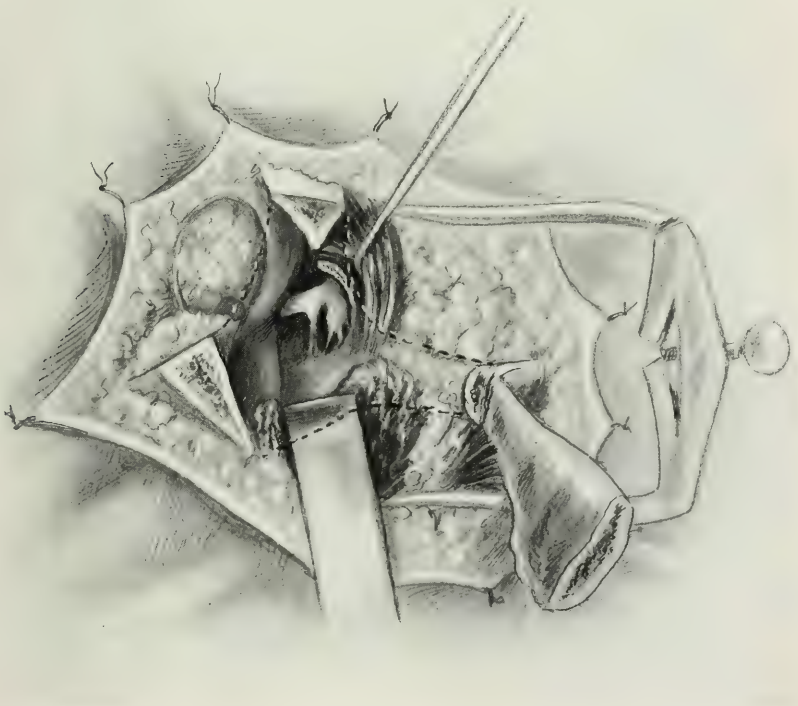


FIG. 8.—I. The internal pterygoid muscle is displaced upwards. II. Pterygoid plate has been removed in part with rongeur forceps. III. The sphenopalatine ganglion and the maxillary division of the trigeminus are exposed to view.

Throughout the operation one does not see the internal maxillary artery. One might have anticipated troublesome hemorrhage from this source, but such is not the case. The only arterial trunk that one sees is the continuation of the internal

maxillary artery in the infraorbital artery. The space in which one works is comparable in size to that in the approach to the

FIG. 9.—Photograph of patient after the author's operation for exposure of the sphenopalatine ganglion.

Gasserian ganglion, and I have found that my illuminated retractor, so satisfactory in the Gasserian ganglion operation, amply illuminates the field.

JAUNDICE FROM PANCREATIC DISEASE*

By CHARLES H. MAYO, M.D.
ROCHESTER, MINN.

In considering jaundice due to pancreatic disease the cases of so-called hemolytic jaundice caused by blood-cell pigment stain, a chronic condition in which the spleen is a factor, and the large per cent of the cases of jaundice in which the gall-bladder is small and there are stones in the common duct, so well described by Courvoisier, are eliminated at once, even though the pancreas may be secondarily involved. Facts relating to the mechanics of jaundice may be stated briefly: Bile is formed in the liver and should pass freely through the hepatic duct and the common duct into the intestine. The common and hepatic ducts are joined at their juncture by the cystic duct from the gall-bladder, an added reservoir, which normally contains about one ounce, although it is capable of distention, should occasion require, to a capacity of several ounces. Just why man and some animals have gall-bladders and others do not is problematic, since the exact function of the organ is not yet known. Part of its function evidently is to maintain the stability of tension in the ducts under which the liver secretes bile and protects itself by the exudate of mucus from its lining. Experimentally, bile from the hepatic duct injected into the pancreatic duct is more irritating than bile from the gall-bladder.

In nature no duct delivers directly into a viscus, but by sigmoid bands or by passing for a space between the mucous membrane and muscular layers of the viscus so that internal compression may block from backflow while it interferes but little with

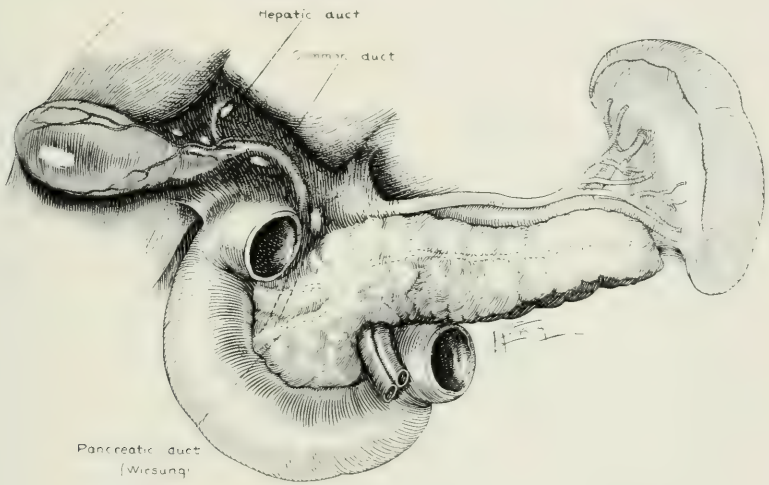
* Read by title.

delivery. It is worthy of note that all tubes or ducts at the point of delivery are closed by muscle rings, while long tubes like the alimentary canal are encircled at each change in structure by automatic muscle rings. Practically all ducts and tubes deliver intermittently through the stimulus of content.

In jaundice bile in the blood causes staining of the skin, conjunctivæ, and tissues, as well as of the urine and unnatural fluids, although the secretion of mucus, milk, and saliva is not affected. The degree of jaundice is comparable with the degree of obstruction. In a study of the manner of the entrance of bile into the blood, Saunders and others experimentally caused jaundice by ligating the common duct of animals; but when the chyle duct is ligated first, lymphatic absorption and delivery into the blood are blocked and jaundice does not develop. The absorption of bile depends on the presence of lymphatics in the liver, gall-bladder, and ducts, and on the pressure or tension. Any cause which interferes with the delivery of bile into the intestine can cause jaundice, for example: Absorption of bile in the liver as a whole or in its parts, caused by new growths, the development of connective tissue with atrophy, cysts or the obstruction of ducts from within by inflammatory and temporary swelling of mucous linings as the result of infection, cholangitis or from acute or chronic diseases, or definite infections, such as malaria, yellow fever, acute yellow atrophy, and toxic jaundice in general; also from within by concretions and parasites and by direct closure from external pressure by enlarged glands or new growths. Anatomically the common duct grooves the head of the pancreas; it penetrates the outer wall of the duodenum, passes indirectly through the muscle and between muscle and mucous membrane for a short distance, and, just before delivering into the free cavity of the bowel, unites, as a rule, with the pancreatic duct of Wirsung; the secretions from the gall-bladder and the pancreas pass through the one outlet.

The area of the pancreas grooved by the common duct is covered by the duodenum by a more or less intimate attachment extending over one-third of the duodenal circumference. The

important feature of this association of ducts is its variability; thus the pancreas may open through the same papilla and the duct be completely separated to the outlet, or the two ducts may unite for a considerable distance as one duct. The two ducts, the pancreatic and biliary, may have separate openings, as in many of the lower animals. The usually larger duct of Wirsung may be the smaller and the duct of Santorini opening between the pylorus and the common duct may be the larger. The two pancreatic ducts may not communicate in the head or



Distribution of lymph nodes along the course of the cystic, hepatic, and common ducts.

in the body of the gland. In considering the relationship between the function of the liver and the delivery of bile in association with the function of the pancreas and the delivery of its secretion, is it possible that the union of the two ducts renders the pancreas more vulnerable to secondary infection following infection of the gall-bladder and ducts? The function of the liver is essential; man could live but a few hours without it. Bile is a by-product of great value of the liver function; probably it provides the system, by way of the intestine, with a constant

immunity against the temporary dangers that may arise from food and its accidental toxins. It emulsifies fats, lubricates the intestine and favors peristalsis; its presence is noted by the color it gives the stool. We have observed several cases of jaundice for from many months to several years in which there was no bile in the intestine; the retention, then, of bile is of itself not a cause of death. We have observed the delivery of all the bile to the surface through a fistula for many months or several years; its retention or delivery into the intestine therefore is not necessary to life, not but that such conditions cause serious discomfort.

The condition or conditions which cause jaundice are of great importance in determining methods of treatment and prognosis. Inasmuch as the pancreas partially or completely surrounds the common duct, any case of jaundice, until differentiated, may be considered as possibly caused by the constriction of the latter. Temporary inflammation, sclerosis and chronic disease or new growths may be the cause. In a fairly high percentage of cases the pancreas is involved by varying degrees of inflammation associated with infection of the gall-bladder and ducts; such a condition and the size of the lymphatic glands and the common duct should be recorded in all cases of operation on the gall-bladder and duct for future reference in case of recurrence of the disease.

The pancreatic secretion is of far greater importance than bile from a digestive standpoint and two ducts were provided for its delivery. It is evident from reviewing the diseases of the pancreas and the surgical procedures developed for their relief that a small part of the normal secretion is sufficient as a hormone or activating agent to give very satisfactory digestive results. Within the pancreas are those very important areas of tissue, the islands of Langerhans, which largely control carbohydrate metabolism. Inflammation of the pancreas is not uncommon; it should undoubtedly be considered a chemical change caused by toxemia from alimentary intake in acute conditions, while subacute and chronic inflammations, which

also are chemical in their effect on the tissue, should be looked on as the result of microbic action. From my experience in operations on the gall-bladder and ducts and examinations of the pancreas I believe that most of the chronic diseases of the pancreas and a few of the acute diseases are secondary to infections of the gall-bladder and ducts, and that a small group of cases of serious acute and chronic infections which cause symptoms do not follow primary gall-bladder disease, but that the conditions are reversed.

Disease of the gall-bladder is now generally admitted to be the result of chronic infection of its tissues, with possibly, but not necessarily, some secondary infection of its contents. The empyema of total cystic duct obstruction is not caused by the same germs that caused the original cholecystitis. The former conception of infection of the bile and then of the gall-bladder from infection reversed through the common duct, or the lymphatics, or the portal circulation by way of the liver, is less plausible than that infection is a direct result of attack through the blood by microbic infarctions in the capillaries. While it is admittedly possible that the pancreas can be involved secondarily through the duct association, especially if bile can enter the pancreatic duct, it is more probable, according to modern investigations of routes of infection, that it also occurs through the blood. The more diseased the gall-bladder is found to be on exploration the less is the probability that the pancreas is the cause of the trouble, and if it is involved the involvement is secondary; the reverse is true with great distention of the gall-bladder and an unobstructed cystic duct. The usual types of pancreatitis are: (1) The intralobular, in which the inflammatory process chiefly involves the outer part of the lobules, causing a lumpy irregular enlargement of the head or of all of the gland, and only in gross disease involving the islands of Langerhans; (2) interacinar pancreatitis in which the surface is smoother and the enlargement of the gland greater, and in which the diffuse inflammation more often involves the islands of Langerhans and is more commonly associated with glycosuria (the latter type

often produces abscesses, and not infrequently is seen with fat necrosis); and (3) hemorrhagic pancreatitis, which occurs as an acute condition of the abdomen, associated with acute inflammation of the gall-bladder and ducts with acute jaundice; but it is a secondary disease from bile in the pancreatic ducts, apparently rendering parts of the gland subject to autodigestion. The pain in some cases of so-called gall-bladder disease may be attributed in part to pancreatic disease, as certain characteristics of pain from pancreatic colic are noted in the absence of the gall-bladder and without obstruction to the flow of bile. Inflammation of the pancreas may occur with dull pain more or less constant in the region of the pancreas, usually attributed to the stomach; the pain is deep, extending to the spine; it may be to the right of the midline in the region of the gall-bladder as well as to the left in the region of the body and tail of the pancreas. With pain may occur an intermittent diarrhea and loss of weight, followed later by jaundice, or jaundice may appear early in a rapidly progressing case; it is probable that in such cases the the common duct deeply grooves the pancreas, favoring early obstruction. Such variations of symptoms are noted with cancer of the pancreas.

A large cystic gall-bladder with the stone blocking the cystic duct and protruding through and blocking the common duct is a rare but possible finding on exploration and is one of the few exceptions (Courvoisier) from the usual small gall-bladder noted in cases of common duct stone. The result of obstruction of the common duct from the pancreas, aside from jaundice, is pain, indigestion, loss of weight and strength and occasionally diabetes. The stools are light colored from lack of bile or from the presence of an excess of undigested fats. Chills and fever are not the rule as with infection and gall-stones. Physical examination in the early or acute condition usually reveals but little; later an indefinite sense of a deep tumor mass may be felt and often a round tumor to the right of the midline is noted, which is probably the distended gall-bladder; the liver is enlarged and, if irregular and lumpy, is probably involved by metastasis.

Cancer of the pancreas is usually not rapid in its progress; it extends over many months, and in the early stage jaundice may vary in intensity as bile passes through the duct from time to time.

In milder cases of chronic pancreatitis there is some difference of opinion as to whether the gall-bladder should be removed or drained for a long period. Mann's research work shows that removal of the gall-bladder greatly lowers the tension within the duct, reducing the spastic closure of the sphincter of Oddi. External drainage of the gall-bladder for a period of many weeks may be justified in certain inflammatory conditions of the pancreas. These conditions but rarely cause jaundice for more than a brief period and of mild degree. However, admitting the difficulty of diagnosis, the greatly distended gall-bladder with jaundice in middle or old age is more probably associated with malignant disease of the pancreas, and many of the symptoms of discomfort are due to the jaundice, the itching, the blood changes and the loss of bile from the alimentary tract. In such cases cholecystoduodenostomy or cholecystogastrostomy, according to the ease and safety in which either may be made, the former generally being preferable, short-circuits the delivery of bile, gives great relief, and undoubtedly prolongs life. The operation is not advised at a time when hemorrhagic ecchymosis from intense jaundice is noted over the body. Some patients with greatly delayed coagulation time of the blood require special preparation and calcium treatment for a prolonged period and possibly blood transfusions before and after operation in order to control the serious complication of hemorrhage, which may even occur from the nose and mucous membrane of the alimentary tract. The fistulous opening remains patent only when the obstruction of the common duct is complete and permanent. The relief is most marked in inflammatory disease, and yet life is greatly prolonged and rendered more comfortable in cases of cancer of the pancreas.

SURGICAL MANAGEMENT OF GASTRIC ULCERS*

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The surgical treatment of gastric ulcer is characterized by a striking uniformity of results. Averages based on statistics from scattered sources show that satisfactory results are being obtained in from 70 to 80 per cent of cases of chronic gastric ulcers in which operation is performed. This average is considerably higher if we include patients whose distress has been alleviated only, so that the number failing to secure any benefit by surgical management does not exceed 5 to 10 per cent. It is this small percentage of absolute and relative failures, however, that the internist emphasizes and that the surgeon regards as a stimulus to increasing the efficiency of surgical methods.

Certain lines of investigation and action could be followed profitably in the endeavor to make the surgical treatment of gastric ulcer even more effective. The part of focal infection in the etiology of the disease is of predominant importance, but a systematic, thorough search for an infectious focus has rarely been included in the surgical management. It is true, also, that the excellent results which follow operation for chronic gastric ulcer have been accomplished with little attention to postoperative dietary or therapeutic management, although it would be logical routinely to carry out an intelligent regime of diet and habits of living. Since these two unquestionably important phases in the treatment of gastric ulcer have been neglected almost entirely in the past, it seems safe to predict that with their full development the number of failures to secure a complete permanent cure will be still further diminished.

* Read by title.

The point I wish to discuss in this paper, however, and one which I believe is of great immediate importance, concerns the selection of operation in cases of gastric ulcer. It is very probable that further progress toward maximum good results in this disease will come from the most intelligent selection and application of the methods of treatment which are now known, rather than from additional operative methods. It has become a platitude that the surgeon should have no fixed ideas of the operation he will perform in gastric ulcer until the lesion is revealed. The recent plea by British and European surgeons, notably Moynihan, for more frequent employment of partial gastrectomy for gastric ulcer, indicates how unsettled is the question of the most efficient use of the various surgical methods of dealing with the lesion.

The observations in this paper are based on the results of the surgical treatment in 826 cases of gastric ulcer in the Mayo Clinic between January, 1913, and January, 1920, and they are intended to point out, as specifically as possible, the relative merits of surgical measures of proved value and some of the indications for their selection.

There are four main considerations upon which the value of any operation for gastric ulcer should be judged: simplicity, applicability, immediate results, and ultimate results.

Simplicity. The simplicity of an operation depends on the ease with which it is performed and the soundness of the principles on which it is based. The popularity of gastro-enterostomy is due largely to the fact that it excels in these two respects. Under ordinary circumstances the operation is simple and safe, and its chief purpose, affording rest to the stomach, is fundamentally sound. The value of the relative rest which is brought about by gastro-enterostomy has not been sufficiently recognized, and yet it is probably the most important function of the operation. Absolute rest of the stomach can be secured by a jejunostomy, and Moynihan advocates and employs this procedure combined with gastro-enterostomy in "Y" for very large fixed ulcers in patients in such poor condition that all other procedures are contraindicated. He reports excellent results of such manage-

ment. The relative rest afforded by a gastro-enterostomy alone, however, seems to be quite sufficient, in all but exceptional cases, to create conditions favorable to the healing of the ulcer.

Another operation which stands out prominently because of its ease of performance is cautery excision; it also is based on sound premises, namely, the effect of heat on infected and malignant tissue. Inasmuch as it is now generally recognized that the best treatment for chronic gastric ulcer must include its radical removal, combined with some procedure to insure symptomatic relief and to prevent recurrence of the ulcer, it is fortunate that we have at hand such simple, safe, and effective measures as cautery excision and gastro-enterostomy, the combination of which has been attended by such satisfactory results. Compared with these procedures, partial gastrectomy, particularly when combined with gastrojejunostomy in "Y," is primarily at a serious disadvantage, for the chances of mishap during its performance are too many to warrant adoption of the operation by any but the most skilled and experienced surgeons.

Applicability. No operation compares with gastro-enterostomy in its applicability, for often it can be employed when all other procedures are contra-indicated, as in an extensively indurated, adherent ulcer high on the lesser curvature, for which resection is not feasible, excision unwarranted, or even destruction by cautery not easily carried out. Not only can gastro-enterostomy be performed in such cases, but the results may be expected to be satisfactory in a large proportion of cases. The wide applicability of the operation and the likelihood of its bringing about good results have naturally led to its being performed when conditions definitely contra-indicated it and especially when no ulcer existed. The extent of its abuse may be surmised from the fact that it has been found necessary to cut off a number of gastro-enterostomies in the Mayo Clinic because the operations had been performed unwisely. When the operation is indicated there are fewer obstacles to its performance than to any other procedure. Even when the stomach is small and high-lying, and cannot be easily approximated

to the jejunum, the anastomosis can be accomplished without undue difficulty. During the last seven years gastro-enterostomy has been a part of the surgical management in 82 per cent of the operations for gastric ulcer in the Mayo Clinic, excluding gastric resections.

Immediate Results. One of the most remarkable accomplishments in the surgical management of benign lesions of the stomach has been the steady reduction of operative mortality. The elimination of former dangers directly associated with the operation, namely, hemorrhage, sepsis, and mechanical disturbances, has been largely responsible for the present low average mortality for all types of operation in all gastric ulcers, acute, perforating, and chronic, 2.99 per cent. This percentage has varied but little during the past five years, so that even with more effective preoperative and postoperative treatment and more careful selection of operation such improved methods affect the general mortality rate by only a small margin. The low mortality rate of 1.85 per cent in 213 cases of cautery excision and gastro-enterostomy in the Mayo Clinic during a period in which the indications for this operation were not so well known as they now are, is significant. On the other hand, partial gastrectomy for ulcer has been associated with a considerably higher mortality than that following knife or cautery excision and gastro-enterostomy; but it should be stated that it has been the practice in the Clinic to resect the stomach only in cases of ulcer with serious complications or those suspected of malignancy. All operative procedures for gastric ulcer are so perfected now that the immediate convalescence of patients is rarely disturbed and serious complications, such as hemorrhage, vomiting, and mechanical difficulties at the anastomosis, have been practically eliminated.

Ultimate Results. It is chiefly in relation to ultimate results that the plea for more radical surgical methods than gastro-enterostomy is made, and the question is mainly whether or not the disadvantages of gastric resection, particularly its greater difficulty and its higher immediate mortality, are out-

weighed by any superior ultimate results which may follow the operation. Ultimate results may be considered under three headings: (1) relief of symptoms; (2) protection against recurrence of the ulcer or occurrence of a new ulcer; and (3) protection against the development of cancer.

Relief of Symptoms. It is fortunate that the results of operation for gastric ulcer, so far as the relief of symptoms of which the patient complained is concerned, can be ascertained readily and accurately without reëxamination of the patient. Certain points in connection with such results may be emphasized: (1) Experience has shown that if a patient has had complete relief for a year after operation it is very unlikely that symptoms will recur, so that statistics and observations based on results after at least eighteen months are relatively accurate. (2) A point to which I have drawn attention in a previous paper,[2] is that some patients, although completely relieved of their symptoms, may suffer from gastric hemorrhages at a later period, this tendency being greatest in patients who have reported hemorrhages previous to operation. It is imperative, therefore, to deal radically with this bleeding type of ulcer, and since we have made a routine practice of destroying every ulcer, duodenal as well as gastric, associated with a history of bleeding, the incidence of these unexpected hemorrhages has been reduced. (3) Of the patients who did not obtain a completely satisfactory result from the operation very few have not been accorded a greater and more lasting measure of relief than they had secured from any previous medical treatment. It is not difficult, if one seeks to find evidence that will appear unfavorable, to obtain statements from patients that they occasionally have some gastric disturbance following operation, which on fair consideration has no more significance than have similar disorders in persons in normal health. Such disturbances are cited, however, as evidences of surgical failures by those who endeavor to discredit surgery or to discredit a certain type of operation. The final judgment of what the operation has accomplished for the relief of symptoms can safely be based on the opinion of the patient.

Protection Against Recurrence of Ulcer or Against Occurrence of a New Ulcer. Gastrojejunal ulcer after gastro-enterostomy, for example, has been so emphasized because of the numerous papers on the complication that a quite unjustified reaction toward this operation has resulted. It is apparently forgotten that ulcers occasionally recur and new ulcers occasionally form after any type of operation for gastric ulcer. Some known factors which contribute to the formation of ulcers in the line of anastomosis, are the irritation of permanent suture material, unnecessary trauma caused by clamps or forceps, and a neglected hematoma. These are controllable factors, but the greatest advance toward preventing the occurrence of these new ulcers will be, I believe, the routine eradication of all septic foci and the adoption of a proper post-operative dietary and therapeutic regime. The complete protection against recurrence of ulcers in any part of the stomach probably depends largely on the thoroughness of such measures.

Protection Against Cancerous Degeneration. The danger of cancer developing on a gastric ulcer for which gastro-enterostomy alone has been performed has been variously estimated. Kocher placed the figure at 2 per cent, von Eiselsberg at 4 per cent, while Coffey has found recently that in his own large experience cancer actually developed more frequently in cases in which excision was performed than in cases in which gastro-enterostomy alone was performed; the latter incidence was only 1 in 163 cases. Our records show that in 214 cases of simple gastro-enterostomy for chronic gastric ulcer there were 5 patients in whom death from cancer occurred more than two years after operation. It must be remembered, however, that the percentage of late deaths following gastro-enterostomy is higher than the percentage following any other operation. While it is not known how many in the former group die of gastric cancer, it is fair to assume, because of the absence of proof of the true character of the lesion at the time of operation, that a considerably larger number die of cancer than is known. One of the most interesting questions in this connection is whether

gastro-enterostomy alone, for an ulcer which has already taken on early malignant changes, does not, besides healing the ulcer, delay growth of the cancer cells already implanted. There is some evidence to show that this occasionally takes place, but very little evidence to support the view that cancer frequently develops on a really benign ulcer which has been gastro-enterostomized. From all information we have been able to obtain on the subject, we have found that cancer has been known to develop, during a seven-year period, in 33 of the 799 cases of gastric ulcer in which operation has been performed in the Mayo Clinic. These known cases of cancer developed after every type of operation, and, what is of great importance, apparently with no less frequency following partial gastrectomy than following knife or cautery excision and gastro-enterostomy. This whole question of subsequent results in relation to the different operations needs continued study. There is one certain fact, namely, that no operation for gastric ulcer will absolutely assure a patient that he will not die of gastric cancer. Whether the incidence of cancer after any operation which includes radical removal of the ulcer is any higher than the normal incidence of the disease remains to be settled and, if proved, explained.

This review of the relative merits of the various methods of dealing with certain types of ulcers and the attempt to establish indications for the operations, are based on the experience with these methods in the Mayo Clinic, and a study of their immediate and late results.

Ulcers of the Lesser Curvature. Ulcers of the lesser curvature, including those closely associated with the lesser curvature on the anterior or posterior wall, comprise almost 90 per cent of all gastric ulcers. Small ulcers of the lesser curvature, that is, ulcers with craters less than 1 cm. in diameter, when situated in the pars pylorica or pars media, and without extensive induration or perigastric adhesions, have been best managed, in our experience, from the standpoint of immediate and end results, by knife or cautery excision combined with gastro-enterostomy. From the standpoint of operative management cautery

excision has certain distinct advantages over knife excision; and the late results of cautery excision combined with gastro-enterostomy, when the procedure has been wisely chosen, are at least as good as those obtained by knife excision and gastro-enterostomy. If either method of excision has been performed radically, the danger of cancer developing is negligible. The value of heat as a destructive agent to the cancer cell which may already have been developed in the tissues surrounding the actual ulcer, has been well established. For small ulcers on the lesser curvature in positions less easily accessible, excision, preferably by cautery, followed by gastro-enterostomy, is very satisfactory and as a matter of fact is the only radical treatment that can be carried out safely. Gastric resection is certainly not warranted in these small, high-lying ulcers because of its technical difficulties, because the amount of healthy stomach removed is out of all proportion to the size of the ulcer, and because of the efficiency of cautery excision and gastro-enterostomy. It may be doubted whether any method of surgical management of these smaller ulcers, in whatever position on the lesser curvature, will prove to be more successful than excision by cautery and gastro-enterostomy, which give satisfactory results in more than 80 per cent. of the cases, and an operative mortality of only 1.8 per cent.

Ulcers of the lesser curvature with a crater larger than 2 cm. in diameter, with extensive induration, adhesions to neighboring structures and organs, and indications of possible malignancy, must be dealt with quite differently from the small, uncomplicated ulcers, to insure the best possible results. If a large ulcer is near the pyloric end of the stomach, pyloric resection is the operation of choice, unless the lesion is so fixed to surrounding tissues and is in such an acute inflammatory state that its mobilization is unwise. Under the latter circumstances gastro-enterostomy is clearly indicated and it will give excellent immediate results with a fair prospect of permanency. The indications for subtotal gastrectomy become less clear the farther an ulcer of this type is from the pylorus, although as long as such an ulcer is reasonably accessible and can be mobilized safely, gastric resec-

tion is the best procedure. Beyond this point of reasonable accessibility, resection is inadvisable because of the reasons already mentioned, and because of the distinctly additional risk. When the lesion is in the body of the stomach and is associated with hour-glass deformity, resection in continuity is a useful technical procedure which will give good immediate results, but sometimes the ultimate results in our experience have been marred by disturbances in motility due to contracture in the site of anastomosis. Deaver and Pfeiffer have recently reported better results from this operation by adding a gastro-enterostomy distal to the circular anastomosis.

The selection of the best operation for the small uncomplicated ulcers and the large complicated ulcers of the lesser curvature is not difficult. When an ulcer between these two types is met with on the lesser curvature, however, the best operation is not so easily chosen. Again the important question is that of malignancy. There are no hard-and-fast rules by which early malignant changes in an ulcer can be recognized, so that differentiation is not easy or certain; but it is a fact that the subsequent course of doubtful cases shows that errors in interpretation are rarely made and when they are it is much more likely that an ulcer has been erroneously labeled "cancer" than a cancer mistakenly called "ulcer." The danger of a primary gastric cancer or a cancerous ulcer being wrongly diagnosed or unrecognized by the combined findings of a well-qualified internist, a roentgenologist, and a surgeon, I believe to be most unlikely, and it is surprising how few of the gastric lesions diagnosed clinically, roentgenographically, and at operation as benign ulcers, prove to be malignant on pathologic examination following radical removal of the ulcer. The incidence of such cases in the Mayo Clinic has not exceeded 1 or 2 per cent. The method of removing a substantial slice from the ulcer and subjecting it to immediate microscopic examination, while not absolute in determining whether malignancy is present, has proved to be most valuable and is quite justified, providing the ulcer is immediately destroyed by cautery, whether or not malignancy is reported on pathologic

examination. This procedure is carried out routinely in the Mayo Clinic in cases which really are not suspicious of malignancy, adding, of course, a gastro-enterostomy. If, however, any part of the examination raises the possibility of malignancy, if the ulcer is favorably situated for resection, and conditions warrant it, partial gastrectomy holds first place, although the higher operative risk demands that the operation be well justified before it is attempted. The farther from the pylorus ulcers in this borderline group are encountered, the less definite become the indications for subtotal gastrectomy, particularly for the surgeon whose experience has not been large in this field. The best surgical management of these cases is, I believe, gastric resection when proof of malignancy is established; gastric resection, if it can be performed without abnormally high mortality, when there is strong suspicion of malignant degeneration; radical cautery excision and gastro-enterostomy, if the resection cannot be performed without an abnormally high mortality; and, in all other cases, cautery excision and gastro-enterostomy, except in cases of ulcer so near the cardia that access is too difficult, when gastro-enterostomy alone will often give surprisingly good results. In cases of very extensive ulcer the usefulness of jejunostomy should be remembered.

Ulcers of the Posterior Wall. After the lesser curvature the most common site of ulcer is the posterior wall: about 10 per cent of all gastric ulcers occur there, and 75 per cent of these are in the pars media. The surgical management of these is technically more difficult and is not as yet attended by as satisfactory results as follow operations for ulcers of the lesser curvature. Such ulcers usually have extensive craters and are attached to the pancreas, and the patients are often in poor condition. It is a requisite of any operative procedure undertaken for such ulcers that the stomach and pancreas shall be separated if good results are to be obtained. This separation may be accomplished by various approaches, such as transgastric, through the gastrohepatic omentum, or through the transverse mesocolon. W. J. Mayo has recently stated his preference for the gastrohepatic

omentum route as the avenue of approach because it is simplest, most applicable, and most effective in exposing the ulcer and its attachments. The ulcer having been separated and specimens removed for microscopic examination, the edges of the opening in the stomach should be thoroughly removed by cautery, the raw surfaces of the pancreas seared, the opening in the stomach closed, and gastro-enterostomy performed. If cicatrization has developed to a point in which there is hour-glass deformity in the body of the stomach and the ulcer itself cannot be dealt with satisfactorily, sleeve resection combined with gastro-enterostomy deals very adequately with the pathologic condition and with the deformity, although symptomatic relief is not certain. When ulcers of the posterior wall are situated near the pylorus (only 8 per cent of them are in this situation), pyloric resection is the operation of choice.

Ulcers of the Anterior Wall. Ulcers of the anterior wall constitute only 1 per cent of gastric ulcers. If the ulcer is small it can be excised primarily with the knife or cautery and direct closure made, or, if the ulcer is near the pylorus, a plastic operation may be advisable. The pyloroplasty of Finney has a definite application in small, non-adherent ulcers in close proximity to the pylorus, although its value seems to be chiefly in dealing with ulcers on the duodenal side of the pylorus rather than on the gastric side. The operation permits of radical removal of the ulcer and direct inspection of the posterior wall of the stomach and duodenum.

For large ulcers of the anterior wall near the pylorus, especially when they have perforated and become adherent to organs and structures, pylorectomy is justified if the mobilization does not involve too wide a dissection into inflammatory tissue; but it should be remembered that these "pyloric" ulcers are almost always duodenal and hence not subject to malignant changes. The same indications for pylorectomy exist when malignancy is suspected or proved, as I have already pointed out. If pylorectomy is not advisable gastro-enterostomy alone without separation of the ulcer from its attachments or any radical treatment of it,

is the operation of choice. Should a diagnosis of malignancy or probable malignancy be made at operation and it seems unwise, because of the patient's condition or the condition of the operative field, to resect the growth, gastro-enterostomy may be performed as a first-stage operation in anticipation of performing resection as the second stage when conditions are favorable. Nothing illustrates so well what gastro-enterostomy will accomplish as its visible effect on these large, adherent lesions; and experienced surgeons all testify to the fact that when the abdomen is opened for the second stage, these ulcers, supposedly malignant, have occasionally entirely disappeared.

To the few remaining types of ulcers of the anterior wall and the greater curvature the general principles that have been suggested may be applied.

The indications outlined for the choice of operation in gastric ulcers of various types and situations are necessarily somewhat general; but they are based on a careful study of the results of operations for 826 gastric ulcers in the Mayo Clinic during a seven-year period. It seems quite safe to conclude from these results that there is every justification for adhering to the practice of excising, preferably by cautery, the benign chronic gastric ulcer, and combining a gastro-enterostomy with the excision. The results in this series of cases do not indicate that more radical treatment, gastric resection, is a preferable method in any considerable percentage of non-malignant ulcers.

It is encouraging to note in summarizing that we were able to trace 85 per cent of the patients in this series. More than 70 per cent report complete relief, 10 per cent report improvement, and 4 per cent only report no relief.

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WOUND EXCISION AND EARLY RECONSTRUCTION IN THE TREATMENT OF COMPOUND FRACTURES

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THE important relation between wound excision and early plastic surgery was constantly in evidence with those assigned to reconstruction hospital service during the World War. Many badly mangled extremities were saved, with all structures essential to function preserved, but were almost useless because of paralysis, stiffness or contracture. This was inevitable with the difficulties unavoidable in military surgery, but much better results should be expected from similar injuries in civil practice.

Observation of conditions in a number of general hospitals in surgical centers leads to the feeling that we are not profiting by the lessons of the World War and the management of these conditions as we should. Of course, this is not true of all hospitals; but could not many hospital-bed days, much dressing material and time of internes and nurses, to say nothing of valuable lives and limbs, be saved by the adoption of the standard instructions to the resident staff as to wound excision, methods of disinfection and general measures of reconstruction by the surgical services in charge of many members of this Association? Frequently we are inclined to ask, what percentage of industrial and other accidents could have been more efficiently treated by the better surgical teams of war times? Yet such team-work and surgical care is much more readily possible in almost any of our home hospitals than in military practice in time of war. War experience showed clearly that

much of such surgery need not be done personally by surgeons in charge, but could be delegated to well-trained younger men fairly instructed how to carry out the work. The yearly increase in thousands of accidents resulting from modern traffic and industrial conditions makes the most efficient management of such injuries a problem of vital importance, for the numbers as well as in loss of life and function equal or exceed that in any field of pathological surgery.

As urged in a paper published over twelve years ago,[1] the most important consideration in the management of compound fractures is still the wound of the soft parts. "If our wound is aseptic, tetanus and blood poisoning are impossible; bony union and a movable joint are favored; osteomyelitis will not develop; and a useful, if not perfectly normal, extremity will usually be saved." The importance of various measures of reconstruction is equally important. Unless we can save or restore the main nerve or blood supply the extremity must be sacrificed. The importance of preserving the function of tendons and certain joints is almost equally great. The ideal, where possible, is to carry out necessary reconstruction procedures at the time of excision.

With regard to wound excision itself the question arises, were the best methods used where possible during the war, and are they now being used? To help throw light on this question some experiments were done in the laboratories of physiology, bacteriology and histology of Cornell University and New York State Veterinary College. I take this opportunity to acknowledge my indebtedness to Profs. Sutherland Simpson, B. F. Kingsbury and V. A. Moore and their associates, Prof. H. J. Milks and Drs. Carpenter and Hitchcock. The problems which we undertook to solve were as follows:

1. Is it possible to disinfect an extensively infected wound, especially the external wound, thus preventing contamination of the clean excised area during excision?

[1] Radical Conservatism in the Treatment of Compound Fractures, New York State Jour. Med., May, 1909.

2. What is the relative value of various antiseptics (*a*) in killing bacteria, and (*b*) in penetrating lacerated and contused tissue?

3. Is it possible so to stain lacerated tissue to be of material help in outlining the injured from the uninjured tissue?

First. In the attempt to solve the first question as to the disinfection of infected wounds, animals which were used in experimental physiology were first killed with an anesthetic. Extensive lacerated and contused wounds were than made by driving a rusty bolt into the tissues with a mallet, by crushing the extremities in a vise against a rough stone, and by tearing with a large rusty hook. The rusty bolt, hook and stone used in making these wounds were all thoroughly contaminated with a culture of a very resistant spore-forming organism. To attempt to disinfect these badly lacerated, contused and definitely contaminated wounds the following antiseptics were tested: (*a*) Saturated solution of permanganate of potassium (this was used because of its oxidizing value with special reference to the anaërobic spore-formers); (*b*) pure carbolic acid solution; (*c*) strong formaldehyde solution; (*d*) Harrington's solution (1 to 500 bichloride of mercury in 90 per cent alcohol with 2 per cent hydrochloric acid) and dichloramine-T 5 per cent. The wounds were then thoroughly swabbed with one of the above strong antiseptics. The results were as follows:

Wound No. 1. Potassium permanganate. Stone. All three tubes show heavy growths of B. Chauveaui.

Wound No. 2. Carbolic acid. Bolt. All three tubes show growths of organism: No. 1, three colonies; No. 2, nine colonies; No. 3, twelve colonies.

Wound No. 3. Harrington's solution. Hook. Two tubes negative; one tube shows one colony.

Wound No. 4. Carbolic acid. Bone crushed. Three tubes show growths of organism: No. 1, three colonies; No. 2, eleven colonies; No. 3, sixteen colonies.

Wound No. 5. Formaldehyde. Bolt. No. 1, negative; No. 2, one colony; No. 3, heavy growth throughout the tube.

Wound No. 6. Dichloramine-T. Bolt. No. 1, sixteen colonies; Nos. 2 and 3, heavy growth throughout the tube.

These results apparently show the superiority of Harrington's solution and also considerable value for formaldehyde solution and pure carbolic acid, but it seems evident that permanganate of potassium cannot be relied upon as an antiseptic in spite of its strong oxidizing value and dichloramine—also did not give good results.

Second. To determine the extent of penetration of the antiseptic into lacerated and contused tissue, frozen sections of excised tissue were made and examined, which showed that, while the degree of penetration is not great, it was probably sufficient to take care of practically all bacteria in case the infection has not been in contact with the tissues too long, giving the bacteria an opportunity to spread by lymphatic or blood channels.

Third. To determine the possibility of outlining the injured from the uninjured tissue the following stains were used: Strong solutions of acriflavine, methylene blue and hot saturated solution of permanganate of potassium. The distinction between stained and unstained tissue was clear enough with any of the stains to be of great help in dissecting out the injured area, but the permanganate solution gave easily the best results. These experiments with stains confirmed similar ones carried out with medical officers at Ft. Riley and the results of clinical experience in civil practice in staining lacerated wounds, sinuses and fistulous tracts for excision. Anyone who has seen the difference between excision of an unstained fistulous tract and one stained to stand out from the surrounding tissues as clear as a shoestring would not be difficult to convince of the value of tissue-staining in outlining irregular penetrating wounds for excision. These methods I have used and advocated for fifteen years and am certain will prove a help to any not using them who will adopt them.

Local anesthesia if properly used is most satisfactory in the vast majority of cases. It was used in a large percentage of the reconstruction work at General Hospital No. 26, Ft. Des Moines.

Wound excision and reconstruction measures in many cases are very time-consuming, and by the use of local anesthesia the

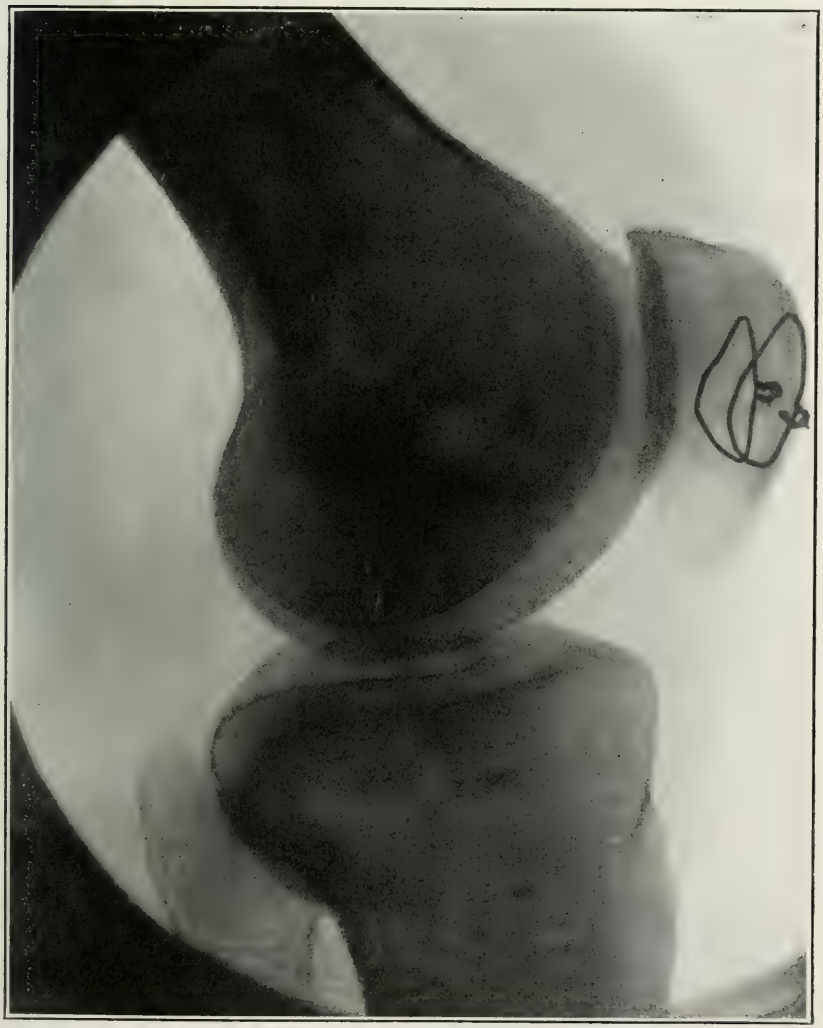

FIG. 1.—Wound excision operation performed 1905. Compound comminuted fracture of patella. Kicked in the knee by a horse. Wound contaminated with horse manure. No trouble from silver-wire sutures five years after operation.

risks are greatly reduced. Crile has demonstrated the value of local anesthesia in combination with gas-oxygen in preventing shock; but, if properly used, local anesthesia alone gives relief from pain which is satisfactory to the patient. As is desirable

FIG. 2.—Perfect flexion of knee, both voluntary and passive, in fractured patella, shown in Fig. 4. Patient able to do all kinds of farm work without any disability.

in any line of surgery, so with local anesthesia, it is necessary to get the best results to study the methods and observe the work of those accustomed to its use. Those who have attempted local anesthesia in major surgery without such study and observation have frequently failed to get relief of pain and discarded

the method without a fair trial. A minority have used local anesthesia for many years and have proved its value beyond

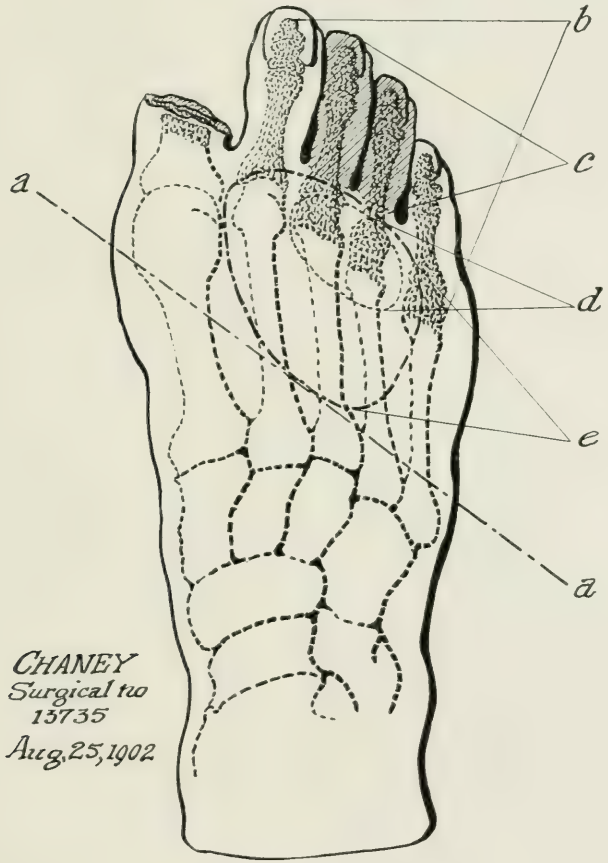

FIG. 3.—Diagnosis: Right foot crushed with railway car wheel. Two middle toes black and gangrenous. *a* direction in which wheel passed; *b*, area of crushed bone; *c*, area that was gangrenous; *d*, area on plantar side of foot where soft parts were entirely destroyed; *e*, area on dorsal side of foot where skin was stripped off and destroyed and tendons exposed.

question in the class of cases which we are now considering. In both civil and military surgery it has the advantage of saving anesthetic drugs and the services of a special anesthetist for the

administration of the anesthetic. In many cases of compound fracture of the extremities it is possible to block the nerves

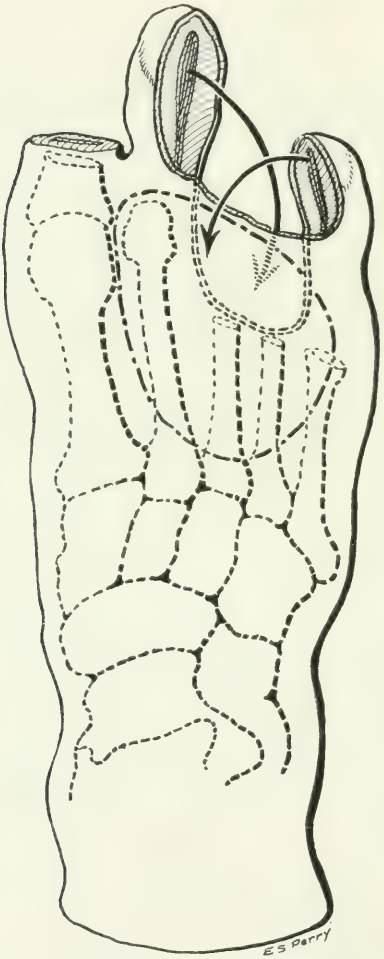

FIG. 4.—Operation: Amputation of first and part of second joint of big toe and of third and fourth toes. Removal of crushed bone of second and fifth toes. Soft part of second and fifth toes saved for flaps.

supplying the field with relatively little anesthetic solution. Both in this way and in certain other cases, by the massive

infiltration, as used by Farr, I have succeeded in getting satisfactory anesthesia. It is true that in certain very extensive and tedious cases the inhibition of the patient partially wears out after an hour or two, but a great deal of time under general anesthesia can almost invariably be saved.

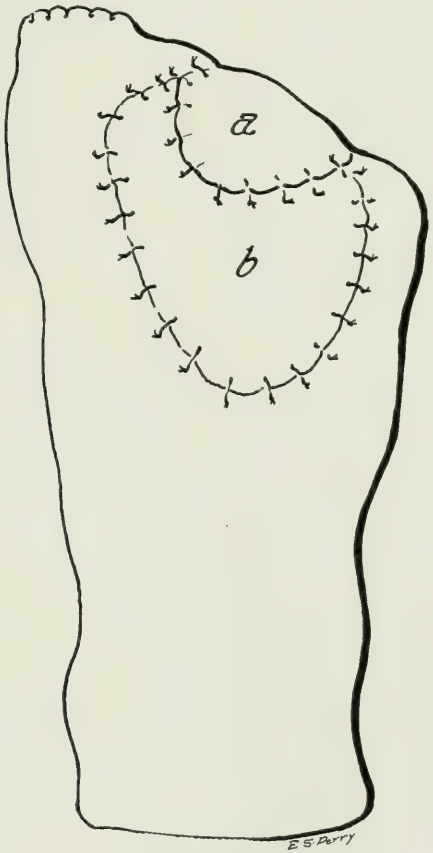

FIG. 5.—*a*, soft parts of fifth toe turned over and across as flap to cover over ends of third and fourth metatarsal bones; *b*, skin graft from thigh over defective area.

Skin Grafts. In the case of superficial injury, when no important structures are exposed, *immediate* or early skin-grafting saves

FIG. 6.—Compound comminuted fracture of humerus. Arm run over by freight car and bone crushed into minute fragments over width of car wheel. Attempt to suture with silver wire unsuccessful, but wire caused no trouble. Bone grafting refused.

a great deal of time in healing. The modified autogenous graft, a name which I coined ten years ago for want of a better,[1] requires only a small superficial denudation which rapidly heals. The use of local anesthesia makes this a very minor procedure in most

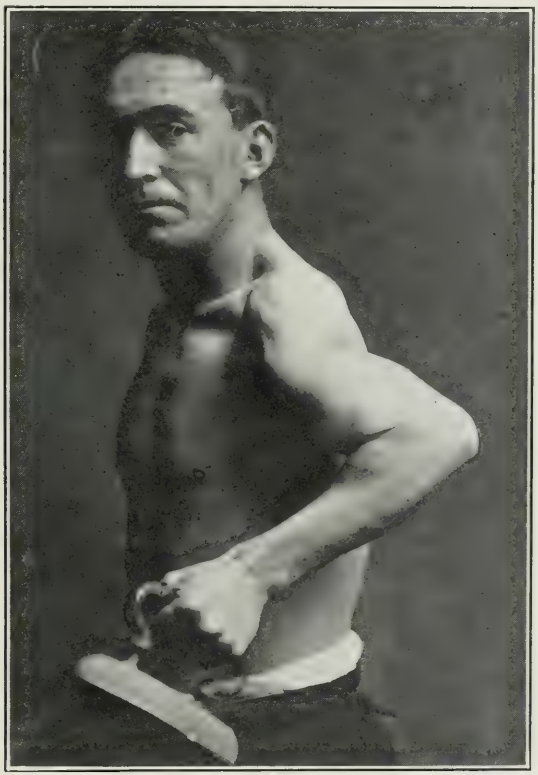

FIG. 7.—Functional result after injury shown in Fig. 6. Flail-joint weakens arm but patient is able to work as freight conductor.

cases, and because of the relatively small amount of skin required it is generally unnecessary to go to distant parts of the body to get skin. The grafts resemble the "deep, small grafts" of Staige

[1] Modified Autogenous Grafting and Turning Skin Flaps to Cover Granulating Surface, Ann. Surg., December, 1911.

Davis, but the method of using them seems to me simpler. A line of skin is infiltrated and the skin pinched up in a roll between the thumb and finger of the operator and assistant. A long strip of skin, thin at the edges but thick in the middle, can then be cut easily with any sharp scalpel and snipped off in pieces

FIG. 8.—Illustrating importance of dealing with wounds of soft parts in case of compound fractures. Patient had a bad compound comminuted fracture following kick by army mule. Complete paralysis of musculospiral nerve which was caught over a small spur of bone in the callus. Patient had complete voluntary extension of the wrist and fingers three months after freeing nerve.

one-half inch or less square. These are supplied as islands about one-quarter inch apart to the surface it is desired to cover and usually grow together rapidly. With free drainage and the use of mild antiseptics the grafts frequently hold even in the presence of considerable infection.

Flaps. When nerve trunks, tendons and joints are exposed, skin grafts do not offer sufficient protection. A pedicled flap of skin with a little underlying subcutaneous fat gives the best

FIG. 9.—Gunshot injury of right forearm with extensive destruction of overlying soft parts exposing extensor tendons; one-third shaft of ulna shot away; about 7 cm. of ulnar nerve destroyed. Illustration shows flaps turned from radial to ulnar side of forearm to cover exposed tendons; autogenous grafts obtained by taking two strips of skin from forearm above, under local anesthesia and snipping into multiple small grafts to cover area from which flap was turned; paralysis of ulnar nerve shown by atrophy of interossei and inability to hold paper by adductors of thumb with right hand. Later at second operation ulnar nerve transplanted in front of condyle of humerus and sutured and pedicle flap from chest to supply skin and soft parts to cover bone graft of ulna, third operation. Good functional arm reported by letter two years after injury.

results. If there has not been too great local destruction of the skin the flap may be turned from the immediate vicinity. In case of extensive destruction of soft parts of the arm, a flap from the chest or, less desirably, the abdomen may be used. Pedicled

flaps turned from the chest or upper abdomen do not seem to be used as frequently as they might be with advantage in the management of injuries with considerable loss of the soft parts of the hand or forearm. A full-thickness flap of almost any size or shape with ample subcutaneous fat can be readily obtained from the chest or abdomen without causing later disability.

FIG. 10.—Pedicled flaps from chest to replace extensive destruction of soft parts of forearm. Greater comfort of patient and adequate immobilization by using binder and heavy pads or small pillows and broad adhesive straps instead of plaster-of-Paris.

The flap becomes well established in about eighteen days, when it can be cut free and adjusted if necessary. As a substitute for plaster-of-Paris or other rigid dressing, the use of wide adhesive strips and small pillows under a binder to support the arm we found in military surgery much more comfortable for the patient, and the fixation all that was necessary for healing-in of the flap.

The slight change of position from occasional adjustment of the binder and very elastic support of this fixation made the patients so comfortable that complaints with regard to the dressing were unusual, in great contrast to plaster-of-Paris. The defect in the chest-wall can be closed with tension sutures of silkworm gut through heavy tubing rubber or something similar, to prevent cutting of the sutures.

Tendons. Extreme disability always follows compound fractures of the hand or forearm in case prolonged suppuration results involving the tendons and their sheaths. If by thorough wound excision or effective disinfection by Carrel-Dakin treatment or other antiseptics it is possible to get fairly early healing, the results of injuries involving tendons are often surprisingly good. In dealing with compound fractures of the finger, hand or arm, an incision along the side following up the divided tendon gives better results than an incision, as so commonly placed, directly over the tendon. With incision directly down through the tendon sheath it frequently springs out and becomes adherent, and in many cases the scar tissue alone gives a high degree of disability. Better still, an incision placed transversely in the line of the normal skin-folds than a longitudinal incision if sufficient exposure can be obtained in this way. Mattress stitches in dealing with thin tendons or muscles usually hold, while simple interrupted stitches frequently cut out and fail to give results. So many failures to secure union of tendons by the use of catgut sutures have come to my notice that I feel that the use of silk or some other unabsorbable material should be urged. In case a tendon has retracted far up, it is frequently possible to reach the end with a Halsted mosquito clamp inserted into the tendon sheath, grasp it and draw it down, a much more satisfactory method than division of the sheath. Splinting or other measures necessary to hold the parts in such position as to avoid tension is desirable but should not be continued long. Usually I begin some movements at the end of a week or two, at the latest three weeks. In case repair is not possible until weeks later, the retracted upper end of the tendon is usually adherent to the

sheath, making the opening of the sheath and separation of adhesions unavoidable. In such cases, bowstring contracture tends to follow tendon suture with considerable resulting disability. Avoiding incision directly down through skin and sheath and early physiotherapy help to avoid this. Tendon-lengthening by various methods has not given me as good satisfaction as direct suture, even where joints have had to be extremely flexed in order to get apposition of the end which had been separated for a long time. In injuries of longer standing the use of free fat transplanted under adherent tendons and covered with a pedicle flap, including a fair amount of fat, frequently give excellent results where there has been loss of function because of adhesions. This is, of course, usually avoidable if reconstructive measures can be carried out early. The value of physiotherapy in mobilizing fingers and joints which have been stiffened is well enough understood not to need more than mention. The importance of tendon repair is most appreciated by persons who use their fingers and hands for highly skilled work. Among my patients have been a number of musicians, including a 'cellist, flutist and pianist, who were completely incapacitated from playing their instruments by reason of relatively trifling injuries involving tendons. These patients have been able to play as well as ever in a short time after suitable measures for repair.

Peripheral Nerve Injuries are common in connection with crushing injuries, gunshot wounds and other causes of compound fracture. The importance of early reconstruction work in these injuries was another of the important lessons taught by the experience of the World War. The records of Dr. E. M. Hummel showed that over 600 peripheral nerve injuries came under observation during nine months following August, 1918, at Ft. Des Moines. Those patients who had had immediate nerve sutures at the front, in several instances, had early return of function. In other instances, partial function was recovered, and in all these cases the secondary operation was made much easier and the prospect of recovery of function greatly improved. Even in case of wounds which became infected the nerve fre-

quently held together, making later reconstruction surgery much simpler. In over 90 per cent of the cases in which peripheral nerve operations were performed local anesthesia was used for an hour or more of the tedious dissection which was usually required to free the nerve and its branches from scar tissue. The patients were made the judges of the efficacy of the anesthesia, and if any complaint was made a general anesthetic was administered at once. The large majority of the patients were entirely satisfied with local anesthesia and the work demonstrated to many skeptical reserve officers the possibilities of local anesthesia in difficult major surgery. Compound fractures with open wounds which came to us infected were rendered relatively bacteria-free within a short time, in many cases, by careful use of the Carrel-Dakin method, making it possible to skin-graft or turn flaps and get rapid and satisfactory healing of the wound and soft parts. Such reconstruction surgery, making possible the restoration of function to muscles and tendons whose movements are controlled by the injured nerves, is equally as important as the repair of the injured nerve. In discussing peripheral nerve injuries elsewhere I have called attention to this important interrelation of function. In a few cases in which secondary operations were necessary we demonstrated to our satisfaction the value of transplanted fat in preventing adhesions and pressure on tendons and nerves. In certain doubtful cases, neurolysis was done, freeing the nerve from surrounding scar-tissue, and in case it was not possible to place it where readhesion was not almost certain to occur the nerve was surrounded by a fat flap. In several of these cases where function did not return within a reasonable time nerve suture was done. In these cases, without exception, the transplanted fat was found in good condition and capable of serving the purpose for which it was used. If nerve function can be restored early a great deal of disability from fibrous changes in muscles can be avoided, as well as tedious freeing of contractures by the various means of physiotherapy. Of course, when contracture or stiffness has developed, the physiotherapy is of great value; but by early reconstruction

surgery such disability usually can be avoided, with saving of time and frequently the difference between a relatively perfect and imperfect result. The autotransplants of Huber (nerve-grafts) have been shown by experience of several observers to give a fair percentage of successes when too much nerve was destroyed to permit of immediate suture by any method. This statement is made on the basis of personal experience and reports of other reliable observers, with all respect for the opinions to the contrary of some good neurological surgeons.

Bone. The rule should be, "Save all that is possible." Army experience showed many useful extremities following infected compound fracture with comminution into almost innumerable small fragments. Carrel-Dakin treatment is of great value if possible to clean the wound by excision without too great sacrifice. Covering exposed bone with a flap immediately or early should be done if possible, but recent experience shows considerable exposed bone may recover under Carrel-Dakin treatment and the fragments form a strong shaft. A fairly useful extremity may be obtained even if a considerable part of the shaft of the long bone is destroyed which is not replaced by bone-graft. Methods of fixation depend largely on choice of the individual, but the army type of splint is well adapted for dressing reconstruction cases. Bone-grafting gave uniformly good results in the hands of many surgeons in the war and it scarcely demands extended comment.

STANDARDIZED RESULTS OF WOUND HEALING

By CHARLES L. GIBSON, M.D.
NEW YORK

This study is an attempt to systematize the records of wound healing by the adoption of a standard which aims at a greater degree of accuracy than usually obtains. It also seeks the causes of imperfect wound healing and to correct such accidents.

Our studies have extended over a period of years and the methods we now employ represent the successive stages of what we believe is a more correct method of investigation. In our early attempts a graphic chart of the weekly results was kept, but later abandoned for a monthly record which more nearly eliminates the statistical fallacies.

The facts and figures which we now present are based on the monthly record from January 1, 1920, to July 1, 1921. They represent only a small portion of our clinical material, as it was thought wisest to stand or fall by a limited group of what might be called ideal cases, that is, presenting no conditions of possible infection from the disease itself. For this record, therefore, all such cases as acute appendicitis, any form of salpingitis, any wound that is drained, are not included in our computation. Originally we graded the operations on the stomach and duodenum, but have not included these in our more recent statistics, for it seemed unwise to include these cases which do not represent the same conditions as the bulk of material, more particularly on account of the impaired nutrition of the patients before operation and during convalescence.

Out of 2277 cases admitted to the first surgical division only

437 were classified as to wound healing. The main class of cases on which grades were given is as follows: All hernia except strangulated; cholelithiasis without drainage; bones (amputations, open reductions and bone grafts); benign tumors (fibroid uterus, cystoma of ovary, dermoid cyst of scalp); laparotomies for malposition of uterus; adhesions of peritoneum; chronic mastitis; varicose veins; chronic appendicitis, etc.

Manner of Tabulating Results. The material is divided into three groups: Grade I represents absolutely irreproachable wound healing; Grade II represents small disturbances of wound healing, such as small hematoma or trivial infection, but none of these accidents delaying wound healing beyond the usual normal period; Grade III represents all other cases, that is, all infections.

We feel it is particularly important to differentiate between hematomata, classified under Grade II, and infections (Grade III), as the Grade II mishaps are more apt to represent individual errors, while Grade III may well be laid to a faulty system.

Manner of Giving Ratings. With rare exceptions the ratings are given by me personally. During my absence in the summer no accurate record has been kept of results. Ratings are made on weekly "field rounds." If wounds are definitely healed, say at the end of seven or eight days, the grade given is usually final. If the rating is given, say at four or five days, a note is given that the final result must be checked up and is so checked up by me the following week.

These notes are all given in the presence of the staff at "field rounds," and if any possible dissension exists the grade is given according to the consensus of opinion. Occasions for such discussion, however, are almost unknown at present, which indicates that our present system is simple and efficient. At the end of the month results are tabulated as indicated on the charts (Figs. 1 and 2).

In addition to the notes made at the weekly rounds, the results of wound healing are discussed at the weekly staff conference which follows weekly rounds. At this conference expla-

nations for disturbances of wound healing are sought and given and decisions are taken which may influence subsequent pro-

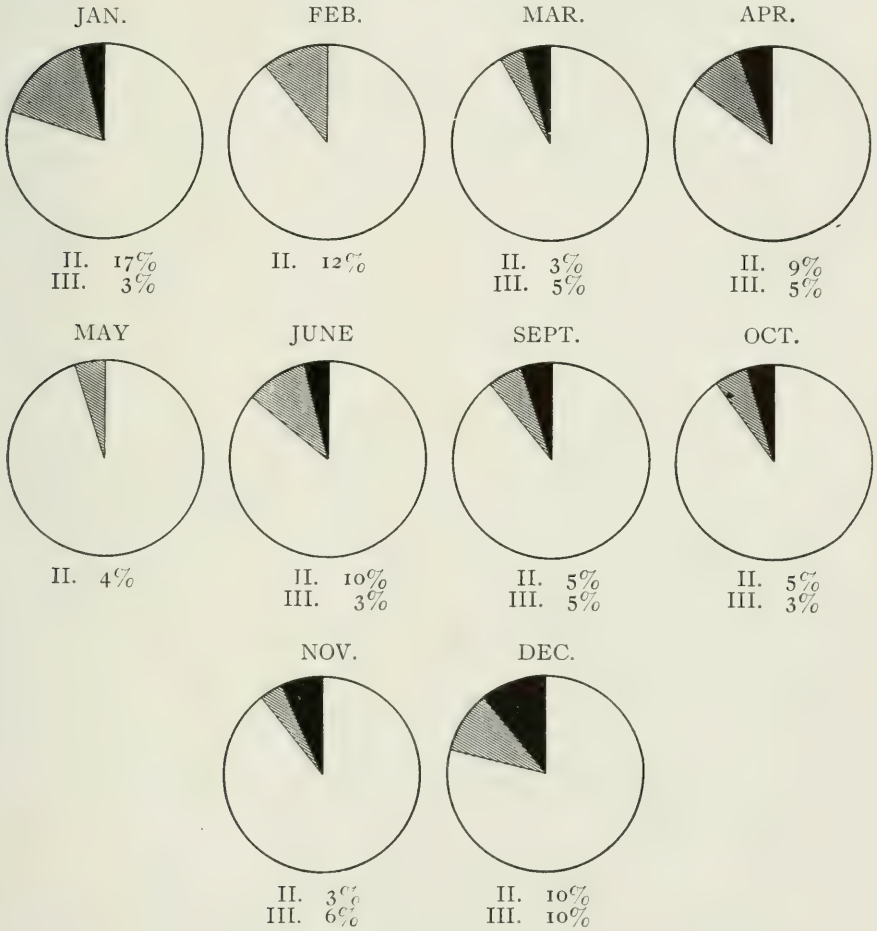

FIG. 1.—Results of wound healing—1920. Class I. (white) Ideal wound healing. Class II. (shaded) Slight mishaps. No detriment to wound healing. Class III. (black) All infections.

cedures. Records are also kept of changes in the staff, both nurses and internes, and the dates of changes of procedures and technic are indicated.

Results. Out of 437 cases classified as to wound healing there were 39 (9 per cent) with disturbances of wound healing. These were divided as follows: Grade II, 25, or 6 per cent; Grade III, 14, or 3 per cent.

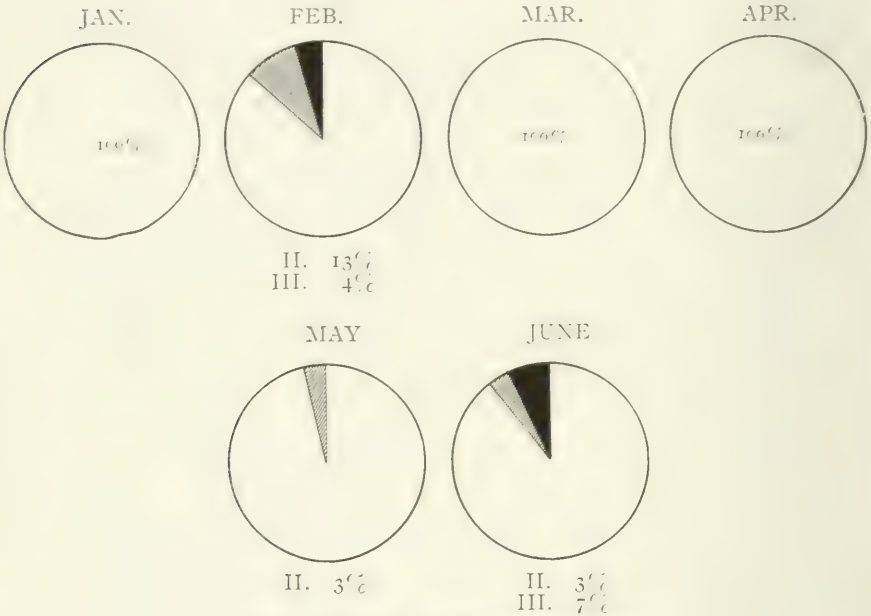

FIG. 2.—Results of wound healing—1921.

The following main classifications are given, showing results of wound healing:

Diagnosis.	No. of cases recorded.	Disturbances of wound healing.	Grade II.	Grade III.
Inguinal hernia	172	14 (8 per ct)	8 (5 per ct)	6 (3 per ct)
Chronic appendix	90	6 (6 per ct)	4 (4 per ct)	2 (2 per ct)
Bone cases	25	4 (16 per ct)	4 (16 per ct)	0
Fibroid uterus	19	1 (5 per ct)	0	1 (5 per ct)
Malposition of uterus	14	1 (7 per ct)	1 (7 per ct)	0
Femoral hernia	13	1 (7 per ct)	0	1 (7 per ct)
Incisional hernia	12	2 (16 per ct)	1 (8 per ct)	1 (8 per ct)
Varicose veins	10	1 (10 per ct)	1 (10 per ct)	0
Adhesions	8	2 (25 per ct)	1 (12.5 per ct)	1 (12.5 per ct)

It will be noted that the main classes are of inguinal hernia and chronic appendicitis. We have long realized that in inguinal hernia we can expect a moderate number of disturbances notwithstanding our constant efforts to minimize them. Of late we have redoubled our energies as regards preliminary cleansing of the skin of these patients, and our latest technical improvement consists in suturing the aponeurosis to the external oblique with interrupted sutures instead of continuous, thereby lessening the danger of disturbance of the circulation and, in addition, turning in the knots of the sutures toward the deep surface of the wound.

An interesting point is in regard to the removal of the appendix in the course of operations for right inguinal hernia. It is our custom to do so when the appendix can be easily brought into view and its removal performed without possible trauma. The following tabulation is rather interesting in that it does not give support to the view that removal of the appendix is a possible source of contamination. This view, I am informed, was held in the army and at one period orders were issued forbidding this procedure.[1]

TABULATION OF HERNIA OPERATIONS. SINCE JANUARY 1, 1917

666 hernia with 33 infections	4.95	per cent
506 hernia without appendectomies—26 infections	5.13	"
160 hernia with appendectomies—7 infections	4.37	"
203 right hernia without appendectomies—14 infections	6.89	"
303 left hernia—12 infections	3.96	"
363 right hernia—21 infections	5.78	"

One case of acute appendicitis with small abscess was removed through hernial wound. Primary union.

One case of abscess base of appendix opened above. Hernial wound healed by primary union.

Other Factors. A study also is being made of the results according to the nationality, age and sex of the patient. So far we

[1] Information furnished by Dr. W. A. Downes.

do not feel justified in drawing any conclusions. We find that at present 80 per cent of our patients are foreign born, and it will be readily understood that, as these patients include many recent immigrants, standards of health, nutrition and cleanliness must be somewhat lower than we would desire.

Preparation of the Skin. Since the fall of 1918 we have replaced tincture of iodine by picric acid, 5 per cent alcoholic solution, and continue to be well pleased with it. We believe our results justify our confidence in its antiseptic qualities and its use has been entirely free from the occasional irritation produced by tincture of iodine. Moreover, we have reason to believe that picric acid, if carried into the peritoneum, causes less damage than tincture of iodine.[2]

In employing picric acid we have no hesitation in using "wet preparation" and the patients are scrubbed and shaved with soap lather. With the exception of hernia, no other form of antiseptic is used.

Conclusions. This paper is not intended to exploit our results and we wish again to insist that the operations here reported do not represent our total series, but merely a limited group of cases which we believe is a fair index of our probable results. What we wish to emphasize is the great importance in a hospital service of carrying out systematically and continuously such an investigation. The psychical stimulus and interest are most pronounced and helpful.

[2] Gibson: The Advantages of Picric Acid over Tincture of Iodine for Disinfection of Skin, Ann. Surg., February, 1919. Farr: Picric Acid in Operative Surgery, Ann. Surg., January, 1921. Gibson: The Results of Operations for Chronic Appendicitis, Am. Jour. Med. Sci., May, 1920.

SOME RESEARCHES ON THE PERI-ARTERIAL SYMPATHICUS

By RENÉ LERICHÉ, M.D.
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The sympathetic nervous plexuses included in the external layer of bloodvessels seem to possess a real autonomy. The systematic study of the phenomena which follow the exciting of average-sized arteries, that is, of prevailing muscular structure, reveals the existence of a very characteristic physiological reaction which never fails in normal circumstances.

A knowledge of the constituents of this reaction is quite interesting from the triple point of view of physiology, pathology and therapeutics; it sets new problems in physiology; it permits us to explain certain pathological phenomena; finally, it authorizes some promising therapeutic attempts.

I feel the best way I can express my thanks for the great honor the American Surgical Association has done me in inviting me to take part in this work is to present to you the results of my researches on this subject these last few years.

I. When you remove the sheath of an artery, the brachial artery for instance, you see that, just at the moment its external layer is pinched, the vessel contracts, its pulse stops at once and its size diminishes. If you excise the cellular layer the diminution will progressively reach the third or the fourth of the normal size of the artery. The segments on both sides maintain their normal size provided the operation has not injured them.

This arterial contraction usually causes the pulse to disappear, but it does not altogether interrupt the circulation.

If the artery is cut a thin thread of blood is seen inside; on the other hand, if during the experiment capillaries are examined by Weiss's method, you will see, at the moment the arterial contraction takes place, the capillary loop diminish regularly in its whole length, pale but remaining visible. If the artery is tied the loop is almost indistinguishable, though a little blood still passes (Fig. 1).

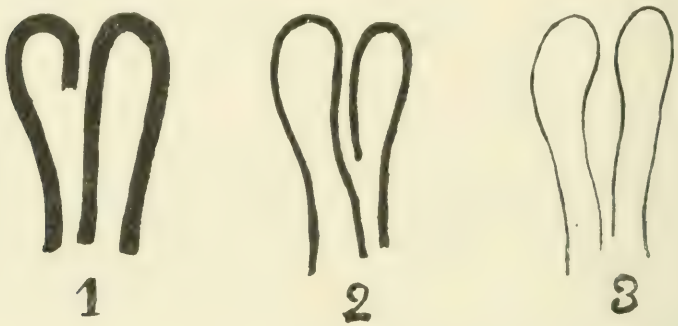

FIG. 1.—1, aspect of a normal capillary loop in a finger, examined by Weiss' method; 2, diminution of size when the brachial peri-arterial nerve is excited; 3, almost complete disappearance after ligature of the brachial artery.

The arterial contraction is the primary element of the characteristic physiological reaction against excitation (Fig. 2).

SECONDARY SIGNS. In the following hours pulse is imperceptible or very feeble; the operated limb colder than the other; there is a difference of 3° or 4° C. After several hours, varying from three to fifteen, secondary signs appear which form the next element of the characteristic reaction.

They never fail when the excision of the nervous layer is really performed; if they are wanting, it is due to the incompleteness of the nervous operation. Out of the sixty-four operations only one failed; in this case, one of my first, the artery was enclosed in fibrous tissue and did not contract during dissection; thus I had not touched its nervous sheath. The secondary signs are the following:

1. An elevation of local temperature reaching 2° and even

3° C., central temperature not being modified. The patient has a subjective sensation of heat. One of my patients complained the day after operation of "a hell fire" in his hand.

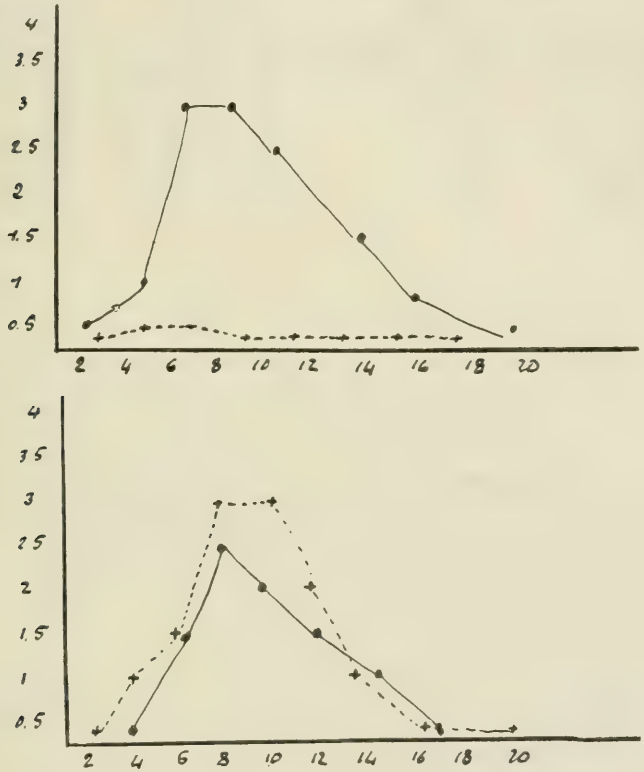

FIG. 2.—1, sudden lowering of the arterial pressure immediately after excitation of the periarterial sympathicus; 2, rise of the arterial pressure following the preceding phases; taken six hours after operation. (____ normal side, _ _ _ _ _ operated side.)

2. An elevation of the arterial pressure, which may reach 4 cm. of mercury compared with the healthy side. Claude Bernard had already noted the same figures when measuring arterial pressure after section of the sympathicus in the neck.

3. An increasing amplitude of oscillations, shown by the sphygmomanometer.

These statements have been verified on my patients by my friend, Dr. Heitz, one of the most eminent cardiologists of France.

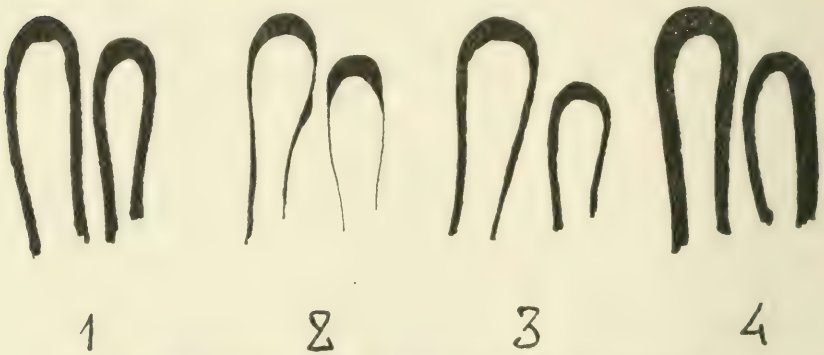

FIG. 3.—Figures showing the capillary loop in a finger and its different sizes before and after Raynaud's disease. 1, normal; 2 and 3, irregular contraction; 4, excessive dilatation following operation.

The vasodilator reaction is transitional; after peri-arterial sympathectomy it becomes attenuated from the fifth to the sixth day and disappears after three or four weeks.

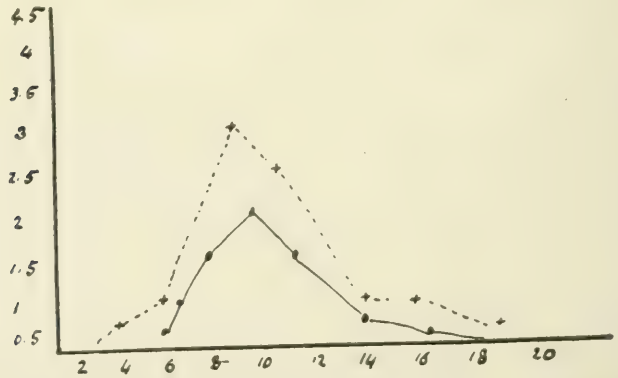

FIG. 4.—Arterial pressure 23 hours after operation. (____ normal side, _ _ _ _ _ operated side.)

Such is the characteristic physiological syndrome of the peri-arterial sympathicus against excitation.

II. Pathologically, this excitation may be provoked as well on visceral arteries as on arteries of limbs, and this by direct traumatic causes or indirect infectious or toxic causes.

Whatever may be the cause, as soon as the peri-arterial sympathicus is injured the physiological reaction appears, in exactly the same way the oculopupillary reaction follows the injury of the cervical sympathicus.

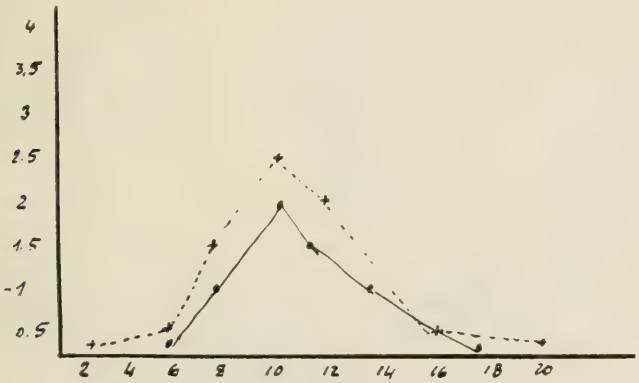

FIG. 5.—Arterial pressure 2 days after operation. (____ normal side, _ _ _ _ _ operated side.)

At the level of the viscera we know absolutely nothing of these reactions; we may suppose that ischemia and prolonged vasodilatation with consecutive elevated blood-pressure may disturb the ductless glands for instance, the thyroid or pancreas, but we have no positive experience concerning the subject. The only experimental fact I know is the following: In a case of diffuse edematous thyroid hypertrophy a unilateral high perithyroideal sympathectomy caused an extreme diminution of size of the corresponding lobe in the next few days. That is a little thing but it allows one to believe there is a very interesting field of future research.

Finally it is only at the level of limbs that we begin to disguish the phenomena resulting from an injury of the peri-arterial sympathicus, and their analysis is not yet easy.

To expose them as clearly as possible one may isolate two groups: In the first group the characteristic physiological

reaction is pure, with its two striking aspects; in the second case, reaction is disturbed and gives various troubles.

First Type. One may choose two characteristic examples, "stupeur des artères" and Raynaud's disease. I will not insist on the well-known syndrome of "stupeur arterielle." It is an active secondary spasm due to sudden excitation of the external arterial layer; a woundless traumatism may produce it as well as a projectile. We have noticed it after violent shock of the humerus, the arm being limp and lifeless, the fingers colorless; one might have thought the artery was crushed. There was really no injury and these phenomena were temporary.

Against Kuttner's opinion I still believe that this contraction is of nervous origin instead of muscular origin, and this for motives I do not want to develop here.

I want only to make you notice that, in certain cases, contracture being so intense may lead to gangrene by insufficient circulation. This led during the war to unnecessary amputations.

Raynaud's symptoms are typical of vasomotor sympathicus disease. In certain cases the peri-arterial sympathicus of the whole limb seems to be excited, for the vascular contraction involves even the capillaries, producing deformations that may be seen on the living limb.

This may be observed surgically when the sympathical irritation is manifest; for instance, in the case of cervical rib or in certain subclavian obliterations.

In these cases the crisis has the exact figure of the physiological reaction with its two characteristic phases, painful ischemia and consecutive vasodilatation.

Second Type. In many circumstances the initial cause is less well known and the physiological reaction is disturbed; either by contracture of too long duration or by abnormally persisting dilatation.

In all cases it produces considerable biological disturbances in the subjacent tissues, creating thus various associations of motor, sensitive, vasomotor, glandular and trophic symptoms, or even signs of local necrosis.

Concerning sensibility there may be no systematized hypoesthesia, but principally painful causalgic phenomena. These troubles are not provoked by an injury of sensitive nerves but begin at the level of the tactile apparatus. The Meissner's, Paccini's and Ruffini's corpuscles are surrounded by a rich vascular net, insuring the perfection of sensations.

FIG. 6.—The forceps catches a fold of the arterial external layer and holds it out of the wound. 1, in both figures the thickness of the external layer has been exaggerated to elucidate the demonstration. (Drawings by Dr. P. Bonnet, associate professor of surgery in Lyons.)

Supposing the vascularization troubled by inflammation, the nervous apparatus suffers at first contact. The sympathic injury producing peripheric vasodilatation has the identical effect; thus may be explained the acute pains in Weir Mitchell's causalgia and certain painful stumps.

By a less-known process, injury of the peri-arterial sympathicus may provoke motor troubles, either stiffness and contracture, a

kind of hypertony localized to certain muscular groups, or a rapid atrophy.

One has noted in "stupeur arterielle" immediate muscular rigidity. The same contracture was also frequently noticed after war wounds and described under the name of reflex troubles by Babinski and Froment.

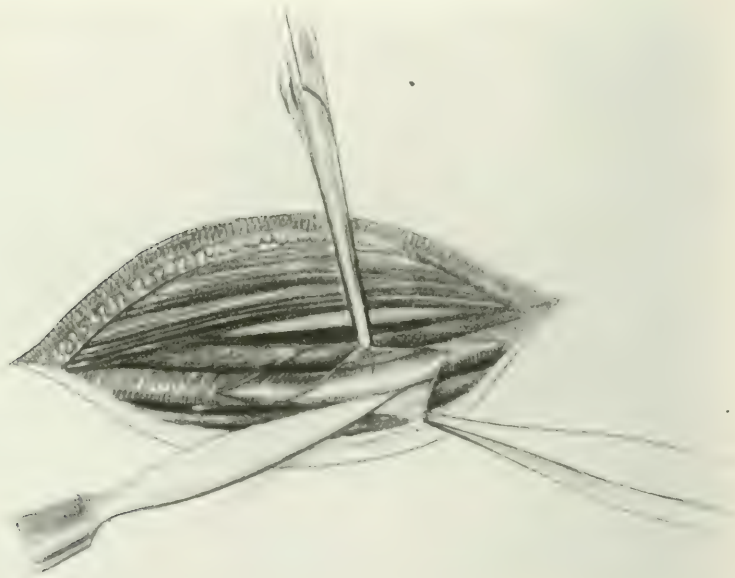

FIG. 7.—The knife opens this fold and softly decorticates the artery.

In these cases when operating on the peri-arterial sympathicus one has seen motor troubles diminish or even disappear as if the contraction of striped muscles were dependent on the sympathicus (Fig. 6).

One may believe muscular symptoms to be but consequences of defective circulation and that sympathectomy acted in modifying it; but one must not forget that the histologists admit for the muscles both a myelinic and amyelenic innervation, and that the influence of the sympathicus on the muscular tonus was

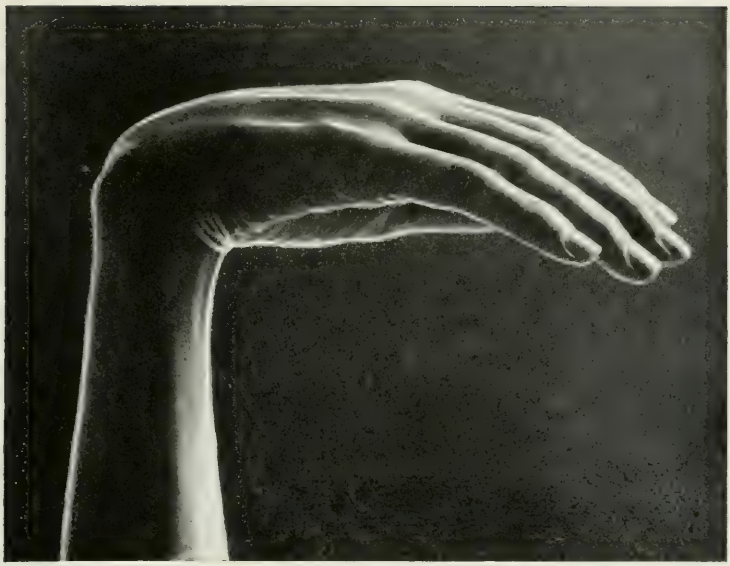

FIG. 8*A*.—Contracture of the fingers after injury of the forearm.

FIG. 8*B*.—Contracture of the fingers after injury of the forearm. Periarterial sympathectomy; result after three days.

shown by De Boer, and the modern neurologists seem to believe that the tonus of muscles at rest is conditioned in invertebrates by the sympathic system. The presence of motor phenomena is thus easily explained.

Connectively one frequently notices changes in the sudoric function, profuse sweating or absolute dryness of the skin, which form a constant symptom of the peripheric feature. As in causalgia, one frequently sees trophic troubles concerning nails, hair or skin and even spontaneous blisters.

Sometimes repeated pressure produces sloughs on limbs whose sympathic functions are disturbed by a traumatism or amputation.

Other vasomotor phenomena are cyanosis, local blue edema or large white edema reaching the origin of the limb without any signs of phlebitis or arterial obliteration. Pains and muscular atrophy may accompany these symptoms as well after deep contusions of the tip of the limb as in small traumatisms.

All these apparently different symptoms have one common point: they are but the manifestation of a circulatory change, vessels being not injured, none of the apparatus concerning them is touched, nor the sensory or muscular systems, or glands or skin. And as all are more or less connected, there is evidently a common origin.

This may not be found in the cerebrospinal nervous system as there is no systematized topography. It is not in the vascular apparatus which is anatomically healthy; operations on the vasomotor system seem to prove that their origin is to be found in the sympathic innervation. In some cases there is an anatomopathological proof: The artery holds more than usually to the common sheath of the vessel by sclerous adherences, and the external layer of the artery is more vascularized. The artery presents a particular aspect, being striped in a parallel way by very fine vessels. I found these lesions with paroxysmal pains in chronic arthritis, in painful stumps and in trophic troubles. But one must know that in most cases one cannot reveal any anatomical change explaining the vasomotor ataxy,

for our knowledge on the anatomopathological features is still very imperfect.

Another explanation of the syndrome might be given: Sometimes when there is a defective vasomotor innervation the causal injury (bruise, fracture, scar) is often distant from the vessels, at the periphery, in a richly furnished zone of sensitive innervation and the vasomotor disturbances seem due to an ortho- or antidromic reflex, starting from the injured point, the way back being the peri-arterial sympathicus.

This is explained by the fact that at the beginning of the accidents the sympathic irritation may be sufficiently intense to bring forth spasm and fatal ischemia in limited parts of the tissues. Thus is produced the local gangrene of Raynaud's disease or after certain prolonged strictures Volkmann's syndrome symptomatic of local muscular necrosis.

Is it not the proof that modified circulation is the real cause of all the phenomena studied above?

III. This leads one to believe that logically the treatment of those vasomotor or trophic troubles must aim to modify peripheral circulation by peri-arterial sympathectomy. The technic is to isolate the artery for 8 to 10 cm., to fix the vascular sheath after dividing, to hold one part with a forceps and dissect cellular tissue either with knife or cannular sound until complete denudation of the vessel, which becomes greatly reduced in size. This brings no injury to the arterial wall. I verified it twice at four and eight months' intervals.

I performed this operation 64 times in: 11 cases of causalgia or equivalent syndromes; 2 cases of painful stumps; 19 cases of post-traumatic contractures; 4 cases of large post-traumatic edemas; 1 case of trophedema; 4 cases of ischemic sequels; 1 case of trophic sloughs on a stump; 10 cases of trophic sloughs after nervous section; 1 case of trophic slough of the heel after medullary injury; 1 case of varicose eczema; 1 case of trophic trouble after frostbite; 1 case of spasmodic paralysis; 3 cases of an attempt to modify tension of cerebrospinal fluid; 2 cases

of Jackson's epilepsy; 1 case of goiter; 1 case of intermittent claudication; 1 case of erythromelalgia.

FIG. 9.—Contracture of the fingers after injury of the forearm. Periarterial sympathectomy; result after six days.

I obtained remarkable successes and had complete failures. Operation failed in the case of intermittent claudication, in one

case of trophic troubles after frostbite, in one case of spasmodic paralysis, and in certain cases of painful syndromes like erythromelalgia.

These failures are easily explained by the fact that the syndrome's nature is still unknown. Sympathectomy was performed, but as an attempt after numerous therapeutic trials. These failures, however, must not discourage, because their analysis may permit one to class in different groups seemingly alike facts and to lighten some syndromes still obscure. This discussion shows the impossibility of delimiting yet exactly the indications of sympathectomy. I only want to give you the results of five years' personal experience.

1. *In painful phenomena* sympathectomy is often very efficacious. In causalgia after war wounds it gave me in 9 cases 2 complete failures, 2 satisfying improvements, and 5 excellent results, 2 of them being noted for several years. Platon has published a series of 12 cases, all seen again after more or less long intervals. He obtained 75 per cent. of successes. In certain painful crises preceding gangrene caused by obliterative endarteritis with or without intermittent claudication it has given the same good results as in painful acroparesthesic syndromes consecutive to bruise of finger, to wounds of hand, palm or footsole. Sympathectomy is thus worth trying in these cases.

It was twice performed for Raynaud's disease and gave a good result.

The result was less constant in painful stumps where I had one success and one failure. It seems to be efficient where the neuralgia of the stump is associated with vasomotor crises, muscular contracture or convulsive cramps. The neurologist Clovis Vincent related recently a striking example: In a case of painful stump, both deep general narcosis and the section of the main nerves failed to attenuate stiffness and the convulsive cramp. Immediately after sympathectomy the muscles relaxed, and forty days after pains had not reappeared.

FIG. 10*A*.—Trophic ulcer of the first toe after section of the sciatic nerve.

FIG. 10*B*.—Trophic ulcer of the first toe after section of the sciatic nerve. Recovery eight days after periarterial sympathectomy.

2. *Concerning muscular phenomena* only the hypertonic symptom is influenced by sympathectomy. Heitz and myself have seen it in contractions consecutive to war wounds. All 18 patients were much improved; the day after operation contracture ceased and voluntary movements became possible. In some very serious cases, only sympathectomy permitted the motor reëducation. I do not believe one can improve by sympathectomy what is wrongly called Volkmann's ischemic paralysis. Whatever may be its starting-point, the syndrome is the consequence of a muscle's focal necrosis; it is a definitive lesion that nothing can modify.

In 3 personal cases of trophedema a rapid diminution of the edema was obtained.

3. *In trophic troubles* leading to ulcers, sympathectomy is very efficacious: 12 cases out of 13 were followed by rapid healing, but relapse is possible if the cause of the trophic trouble has not been removed, and the cause is not always removed by the sympathectomy.

This is the case of perforating ulcers which never were operated successfully and therefore after my experience I should not advise it. Sympathectomy was also wanting in a case of numerous ulcerations consecutive to frostbite. The initial lesions, neuritis or arteritis, provoked relapse of the ulcers.

4. In cases of trophic symptoms after nervous sections results were excellent. For 10 patients I obtained rapid healing of the ulcers by sympathectomy. However, after observing 17 cases, I believe sympathectomy should be replaced here by the removal of the neuroma of the sectioned nerve, which is the starting-point of vasomotor reflexes causing the trophic trouble. The removal of the neuroma must be followed by the reconstitution of the nerve's continuity either by suture or by grafts, avoiding thus the relapse of the neuroma. I performed 7 grafts with complete satisfaction and now prefer this method to sympathectomy in spite of its satisfactory results.

What is the real action of sympathectomy? How does it realize healing of the most desperate ulcers? This seems to me

due only to local circulatory hyperactivity noted always after sympathectomy in the following hours, vasodilatation being apparently one of the optima causes in the tissue's growth;

FIG. 11*A*.—Spontaneous ulcer of stump with vasomotor troubles.

FIG. 11*B*.—Spontaneous ulcer of stump with vasomotor troubles. Healed by periarterial sympathectomy.

certain local giantisms observed during growth depend probably on constant vasodilatation. After sympathectomy, vasodilatation accompanies local elevation of temperature and of pressure, both favoring healing. This fact is experimentally proved in the following way: Having produced vasodilatation in a rabbit's ear by removal of the superior cervical ganglion, one practises on both ears a wound of equal size. The sympathetized side is more abundantly bleeding and more intensively red colored. Four days later its wound is covered with granulations, epidermis begins to grow, while on the opposite side the wound remains gray-colored without any epidermic border. On the eighth day there is complete healing on the sympathectomized side, while on the other side healing extends to half the wound. A fortnight later the scar of the vasodilated side is more regular and supple, of better quality than the other and shows no retraction.

Peri-arterial sympathectomy has identical effects. Healing of trophic troubles is thus a question of vasodilatation, and therefore peri-arterial sympathectomy must be tried in these cases. The characteristic of sympathectomy is to be more vascular than a nervous operation. For this reason its consecutive vascular reactions might perhaps be employed in many circumstances. Producing an immediate ischemia, lasting eight or ten hours, one can perform it to insure preventive hemostasis. I did it successfully in three brain operations. On the other hand, the postoperative vasodilatation being lasting, it could be utilized to help the insufficient circulation, for instance, in obliterative endarteritis, or to prepare the reëstablishment of cerebral circulation previously to ligature of the common carotid. Therefore in spite of the hypothetic character of it I must believe the question ought to be studied concerning the viscera and glands. I wonder if the possibility of producing locally active hyperemia might not give us a new power of action on organs when these are becoming insufficient? Only repeated experimental researches may reveal to us what those suggestions are leading to—they are not yet begun; I offer them to your appreciation.

DISCUSSION ON PAPERS OF DRS. TINKER, GIBSON AND LERICHÉ

Dr. Harvey Cushing, Boston:

It has been a privilege for this Association to hear this most interesting account of the researches in which Dr. Leriché has been engaged. There has been an effort for a long time on the part of surgeons to make operative use of the sympathetic system in one way or another in combating certain symptoms, and we all are familiar with the early operations on the cervical ganglia for facial neuralgia, for thyroid disease and for various other conditions. When this ganglion is removed for persisting exophthalmos we know that in some cases it gives fairly satisfactory cosmetic results, but causes so many other disturbances it has been abandoned.

Dr. Leriché has taken this difficult subject up anew, and whatever the future may have in store for this procedure, certainly the evidence of prompt healing shown in Dr. Leriché's very remarkable pictures must make us all feel that the method is something we must at best not ignore.

As Dr. Leriché admits, the matter is in the experimental stage and it is a matter of encouragement that so many of our laboratory workers, who are perhaps more interested in these things from a physiologic standpoint than from the surgical application, are engaged in similar studies. I refer principally to the work of Cannon. All of these things show the current trend toward some therapeutic adaptation of the knowledge which Langley and his school have given us concerning the function of the sympathetic nervous system.

There is one thing that I think could be utilized in connection with these operations of Dr. Leriché, namely, the tourniquet, for by its use one may determine whether these measures he has described are likely to have the looked-for beneficial result. All of us are familiar with the effect of the tourniquet in causing a temporary paralysis of the vasoconstrictors which leads, particularly in amputations, to what is called a wet stump. All are familiar, too, with the temporary paralysis which follows a prolonged application of too tight a tourniquet. These facts led me some years ago to utilize a tourniquet in cases of Raynaud's disease, and I have had a considerable number of patients who felt that they were much benefited by the daily application of a tourniquet. One of these patients was a seamstress who, particularly in cold weather, was unable to handle her needle, owing to vasomotor spasm which caused "dead fingers." But she learned to use a rubber tourni-

quet which served to produce the secondary flush, which we are familiar with in amputation stumps, and this flush would persist for some hours or even for the remainder of the day. I have seen the method in other cases save fingers from actual necrosis. These procedures with a tourniquet are more conservative than those advocated by Dr. Leriché, which consist of actual severance of the nerves accompanying the vessels, but they lie in the same direction.

Just how long after severance of the nerves the vasomotor paralysis remains he has not told us. It certainly cannot be long, for sympathetic fibers reunite with great rapidity. The point I wish to make is that in some of his cases it might have been possible to determine whether the condition was likely to be benefited by a sympathectomy if the tourniquet had been systematically employed for a time beforehand to see whether the condition was ameliorated.

I rose, Mr. President, largely to thank our guest, Dr. Leriché, with whom many of us are well acquainted, for presenting his valuable paper before this association.

Dr. Howard Lilienthal, New York:

I would thank Dr. Leriché if he would say something about the technic of the operation, which I did not catch if he did mention it. I should like very much to make use of the method. It may be an explanation for the reported cures following attempted reversal of the circulation in juvenile gangrene, obliterating thromboangeiitis, even though this disease may not be due to sympathetic disturbances.

Dr. John H. Gibbon, Philadelphia:

In regard to Dr. Tinker's interesting paper, it would seem appropriate to mention the use of paraffin in the treatment of granulating wounds. We all are familiar with the remarkable results obtained by the paraffin treatment of burns; during the past winter at the Pennsylvania Hospital I have used this treatment on all superficial granulating wounds with surprisingly good results. The following case illustrates what can be accomplished by this treatment: A woman, who had had both legs severely burned between the knee and ankle, was sent to the hospital for amputation of the left leg, which had never healed. There were a few islands of skin of varying sizes between the knee and the ankle, but the ulcerated surface practically covered the entire leg and encircled it at several points. Amputation seemed inevitable, but we concluded to try the paraffin. After a few applications the discharge greatly decreased and after eight or nine weeks cicatrization was complete. In all chronic ulcers, especially those in cicatricial tissue,

this method of treatment gives the best results I have seen. We are using paraffin also for skin-grafting operations.

Dr. Gibson's report of wound-healing shows that hernia wounds are more liable to become infected than others, an experience which can be readily confirmed in the experience of us all. I believe that these infections are not the result of the situation of the wound or any difficulty in sterilizing the skin in this region, but to interference with the circulation, due to too tightly tied sutures. Too many operators have the idea that as they are endeavoring to repair a breach in the abdominal wall the sutures must be closely placed and tightly tied. This method of repair favors infection, necrosis and atrophy, and, as a consequence, a recurrence of the hernia. A comfortable approximation of the structures with absorbable sutures offers the best and strongest healing.

In regard to Dr. Leriché's paper I feel that he has presented something to us which has passed beyond the experimental stage, a remarkable contribution, and the Association must feel greatly indebted to him.

Dr. Martin B. Tinker, Ithaca, N. Y. (in closing):

As regards the wound-excision cases, the observations of the results in army work have led me to think that there are about four kinds of results:

1. The excisions which were so imperfectly done that they might almost as well not have been done at all—so imperfectly that pieces of clothing and foreign bodies were left in the wounds.

2. Those in which the excisions were so extensive that the patients suffered disability from cutting out structures of vital importance.

3. Those in which the excisions were done satisfactorily as regards the healing of the wound but in which the patient was left with a practically useless extremity, because structures of a great deal of importance were not restored. The reconstruction work could not have accompanied the excision in all cases, but in some of the cases in which it was done at the same time it was very satisfactory.

The combination of the two—excision sufficiently thorough to give good wound-healing and at the same time avoidance of injury of structures of importance to function or restoration of function by repair of these tissues is the ideal.

I should like to join with the others who have spoken of the value to them of Dr. Leriché's paper. We all have met with many of these cases in which we have not known what could be done to improve their condition. I think we are greatly indebted to him for his valuable suggestions.

REPORT OF THE COMMITTEE ON FRACTURES

By W. L. ESTES, M.D., Chairman
BETHLEHEM, PA.

The committee presents its final report. This report is the completion of the 1915 report and may be received as a combination of the preliminary 1915 report with subsequent investigation and later data.

The same general scheme of investigation as was indicated in the 1915 report has been followed, namely:

1. Only fractures of the long bones have been studied.
2. The same regions or divisions of the bones have been noted.
3. Simple and compound fractures have been separately classified, investigated and tabulated.

The four main points of inquiry and investigation have been continued, namely:

1. What is the average present-day anatomical and functional results in both simple and compound fractures in the several age groups?
2. What is the average period of disability of (*a*) simple and (*b*) compound fractures of the several long bones?
3. What is the comparative value of (*a*) the non-operative or closed methods and (*b*) the operative or open methods?
4. What is the comparative success of immediate and delayed treatment in each group of cases?

The blank form recommended by the committee has been slightly changed. It was formally adopted by this Association, recognized by the American College of Surgeons and has been introduced into many hospitals in the United States as the accepted blank for recording fracture data. Its continued and regular use

in the hospitals will result in having available for future investigation adquate records of fractures, and we hope will induce surgeons to see to it that their fracture cases shall always be followed up and the final results accurately determined and noted.

FRACTURE RECORD

American Surgical Association

General Results: GOOD MODERATE BAD

Anatomical

Functional

1. Bone
2. Site—Neck Upper Middle Lower 3d Condyle
   Involving joint
3. Name
4. Sex—M F
5. Age
6. Occupation
7. Time fracture occurred—Date Hour
8. Hospital entered—Date Hour
9. First treatment—Date Hour
10. Cause of fracture
11. Kind of fracture—Oblique Transverse Spiral
    Impacted Comminuted Simple Compound
    Greenstick Subperiosteal
12. Was there serious injury to soft parts—Skin—Yes No
    Muscles—Yes No Vessels—Yes No
    Nerves—Yes No
13. Reduction: How many hours elapsed after accident before reduction
14. Was anatomical reposition of fragments obtained—Yes No
15. Anesthetic used: Yes No Ether Gas
16. Fixation: Closed Method.
    Position—Hyperflexion full supination abduction
    Splints
    Plaster of Paris
    Traction—Buck Thomas Hodgen
    Balkan frame Tongs
    Amount of weight used

17. Fixation: Open Method.
Was non-operative treatment tried first.....
How long after injury was operation performed.....
Was open reduction alone performed
What form of internal fixation used—Steel plates.....Wire.....
Nails.....Screws.....Bone transplants or implants.....
Was it later necessary to remove fixation materials—Yes.....
No..... Date.....
18. Shortening at first examination.....cm.
When all apparatus removed.....cm. Date.....
When discharged from hospital.....cm. Date.....
At last observation.....cm. Date.....
19. Roentgen ray used—Yes.....No..... First finding—Date.....
day before reduction; .....day after reduction

Plate No.	Fragments displaced, date.	Not.	Slightly.	Markedly.	Overriding.	Rotation.	Angulation.
	Before reduction						
	After reduction						
	After union						

20. How long confined in bed..... How long in hospital.....
21. How long did patient use crutches.....Cane.....
22. Results: Final examination made.....weeks.....months after injury.
Union: Bony.....Fibrous.....Non-union.....
23. Disability: Absent.....Partial.....Complete.....
Estimated by.....Shortening.....Angulation.....
Swelling of soft parts.....Pain.....Nerve involvement.....
Interference with joint function.....Endurance.....
24. Mortality—Main cause of death.....Age of patient.....Shock.....
Hemorrhage.....Other injuries.....Sepsis.....Exhaustion.....
25. Duration of absence from work.....weeks.....months.....
26. Is patient fully able to take his former job.....
27. Present wage-earning capacity compared with former.....
28. Compensation under insurance, legislative act or legal process obtained—
Yes.....No..... Expected—Yes.....No.....

The investigation on account of the difficulty in securing reports and the necessity of searching through many incomplete reports in order to find adequate data has proved onerous and trying. In order to lighten the labor and to obtain the best results the committee was divided and a certain part of the work assigned to each as follows:

1. To Dr. J. B. Walker was assigned the investigation and report of fractures of the A. E. F. during the war, with ultimate results.

2. To Dr. T. W. Huntington the fractures of the humerus in civil life.

3. To Dr. Edward Martin the fractures of the bones of the forearm in civil life.

4. To Dr. A. P. C. Ashhurst the fractures of the bones of the leg in civil life.

5. To Dr. W. L. Estes the fractures of the femur in civil life.

Each head of a division will therefore make a report of his investigation of the fractures belonging to his division and should receive full credit for his work.

The following-named Fellows served as the auxiliary committee and rendered valuable service in assisting, collecting and examining cases. Divided into groups a certain number were assigned to each division and assisted in drafting and formulating the reports of the several divisions.

Dr. Walker: Fractures in War. Assistants: Dr. Jones, Dr. Matas, Dr. Starr.

Dr. Huntington: Fractures of the Humerus. Assistants: Dr. Jonas, Dr. Freeman, Dr. Terry.

Dr. Martin: Fractures of the Bones of the Forearm. Assistants: Dr. Thompson, Dr. Ransohoff.

Dr. Ashhurst: Fractures of the Bones of the Leg. Assistants: Dr. Speed, Dr. Gaub.

Dr. Estes: Fractures of the Femur. Assistants: Dr. Graham, Dr. Vaughan.

We gratefully acknowledge their valuable assistance in preparing this report.

First Division. Military fractures, 2083 cases.

Second Division. Fractures of the humerus, 162 cases: non-operative, 117; operative, 45.

Third Division. Fractures of the bones of the forearm, 271 cases: non-operative, 227; operative, 44.

Fourth Division. Fractures of the leg bones, 926 cases: non-operative, 739; operative, 187.

Fifth Division. Fractures of the femur, 654 cases: non-operative, 471; operative, 183.

Altogether the committee collected and studied over 8000 cases of military fractures and 2013 cases in civil life. Of these civil cases 1521 were simple fractures and 482 compound. The results of all these cases have been carefully investigated.

Unfortunately the records of many of the cases were so incomplete that they could not be used in their entirety.

The cases have been tabulated under six principal heads, namely:

1. To show the results in the following age periods, viz.: (*a*) One to fifteen years; (*b*) fifteen to forty-five years; (*c*) forty-five to sixty years; (*d*) over sixty years.

2. To show the effect of good anatomical restitution in shortening the period of disability and restoring full function.

3. To show comparative results in the treatment of simple fractures of the operative (open) method and the non-operative (closed) method in the several age groups.

4. To show the result of the operative and non-operative methods in the treatment of compound fractures, also considered as regards the several age groups.

5. To show the results of treatment of the fractures of the several long bones in the various regions in the several age groups, in order to determine the average period of recovery.

6. To show the comparative results of immediate and delayed treatment of the closed and open methods of treatment in the several age groups.

The operative cases are considered as:

1. Immediate.
2. Delayed.

All operations performed within fourteen days after injury are classed under immediate operations; those done after this period are classed as delayed operations.

The results of operations for non-union and for deformities

resulting from fractures are not included nor considered in the report at all.

The committee finds:

1. The results are best in the age period under fifteen years. Conservative treatment is generally effectual during this period.

2. Good anatomical restitution of a fractured long bone results in the best functional result and has the shortest period of disability.

3. While comparatively few open operations are reported under the fifteen-year-age period it seems to make little difference in the result, except in senile cases (where it is unfavorable), what the age period is when the operation is done.

4. The end-results of non-operative and operative treatment of compound fractures show very little difference in the anatomical result, but the functional results are better after operative treatment, except in compound fractures of the shafts of both bones of the leg; here the reverse seems to be true.

5. The average period of disability (that is the time lost from work) in simple fractures is as follows:

For fracture of the shaft of the humerus	14.0 weeks
For fracture of the head and neck of the humerus	11.5 "
For fracture of the condyles of the humerus	9.0 "
For fracture of the shaft of both bones of the forearm	10.8 "
For fracture of the femur, all sites, adult cases	8.2 months
For fracture of the femur, all sites, children	4.5 "
For fracture of the leg, all sites	4.7 "

(Periods of disability were not recorded accurately in many of the reported cases and very seldom in compound fractures.)

Compound Fractures

For fractures of the femur	11.0 months
For fractures of the leg	7.0 "
For fractures of the upper extremity	4.0 "

6. For good functional results the humerus should show not more than 1 cm. shortening and no appreciable angulation. No pain or paralysis should result.

The forearm bones should show no shortening; function should always be good and no lasting pain result.

Fracture of the shaft of the femur should not result in shortening greater than 2 cm., nor in a fixed position of angulation or rotation. The function of all joints should be good.

Fracture of the shaft of the bones of the leg should result in no appreciable shortening and no angulation or rotation. All function of the joints should be preserved.

7. There is no method or splint universally applicable; all depends upon the discrimination of the surgeon and the manner in which the apparatus is applied and maintained.

The late war has brought into prominence the suspension-traction method for treating fractures. The Balkan frame or the Hodgen splint for suspension is used, and tongs or the Steinmann nail are used for traction directly on the distal fragment. The Thomas splint has proved of great value in the treatment of fractures of the shaft of the femur; it is recommended especially when hospital treatment cannot be obtained.

Plaster casts and molded splints are indicated and are useful only after a fracture has been satisfactorily reduced.

RECOMMENDATIONS. 1. The committee recommends as a general principle that fractures be treated by a skilled surgeon.

2. Roentgen-ray pictures should be made by a competent radiographer and a fluoroscope should be used for diagnostic purposes and for guidance in applying the permanent dressing. At least two skiagrams should be taken, and they should be taken from opposite perpendicular directions. Skiagrams should also be taken after permanent dressings are applied, to prove proper reduction, and at the end of treatment to show the results of the union and for the purpose of a graphic record.

3. Fractures should be reduced immediately after the injury if it is possible to obtain and apply proper retaining apparatus or splints. The statistics show markedly better results when the treatment is begun at once. It is, however, not only useless but cruel to subject the patient to the pain of manipulation for reduction unless the surgeon has proper fixation apparatus at

hand and the subject is where he may have a permanent dressing applied.

4. General anesthesia should be employed, as a rule, to facilitate reduction and to prevent pain, unless the condition of the patient contraindicates it.

5. Neither the non-operative nor the operative method is to be recommended exclusively. Each has its indications and should be employed when required. Generally speaking the age period under fifteen years is the period in which non-operative methods are especially effectual.

6. The open method when adopted should be employed early. It may be used at any age period, except in senile cases, whenever a skiagram shows a deformity or a position of the fragments which obviously cannot be reduced or when proper efforts at reduction and retention have proved unavailing.

7. Some form of rigid plate applied directly to the bone seems to be the best fixation method in operative cases.

8. Open operations for simple fractures should be undertaken only by experienced surgeons who are thoroughly equipped by training and who have proper instruments and apparatus to meet all the possible indications of the operation.

9. After fracture of the long bones of the lower extremity, some efficient form of caliper should be used when the patient begins to walk, and should be continued for some weeks, in order to prevent yielding of the newly united fragments to the weight of the body and the production of bending and distortion at the seat of fracture.

10. The treatment of any fracture ought not to be considered complete until full restitution of function has been secured. For this purpose every hospital which treats fractures should be equipped with apparatus for mechanical, electrical and hydropathic treatment. The reconstructive treatment should be considered in every case an essential part of the general treatment. Also, in order to make the record of every case of major fracture complete a careful follow-up system should be adopted and sedulously followed.

11. The work of this committee has been greatly hampered by the inadequacy of the records submitted for its consideration. A large proportion of the cases had to be rejected entirely, and most of them were so incomplete as to make deductions based upon them misleading.

The first step in the betterment of practice is the study of results achieved by present-day methods. An adequate study is impossible without complete records.

FRACTURES IN MILITARY SERVICE.

By JOHN B. WALKER, M.D.
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Among the 224,089 battle casualties in the A. E. F. there were 27,013 patients, with 18,639 fractures involving the long bones: 5063 occurred in the femur, 4254 in the humerus, 1066 in the fibula, 2679 in the tibia, 1945 tibia and fibula, 1527 in the radius, 1225 in the ulna, 801 radius and ulna, 79 not stated.

The most serious cases were the gunshot fractures of the femur, and it was in just this class that there developed the greatest improvement in the treatment of fractures during the World War. Of the 5063 patients with this fracture, 1010 (20 per cent) died; while in the Civil War, of the 6738 fractured femurs, 3434 (50 per cent) died. Of the above 1010 deaths, 40 per cent died within the first seventy-two hours and 55 per cent within the first seven days.

Treatment. Infection to some degree was present practically always; generally it was severe and persistent. Osteomyelitis became the most serious complication. Those cases in which the primary operative work was thoroughly performed within the first twelve hours made by far the best recovery. In those cases which were operated on after twenty-four hours the percentage of infection and mortality was more than doubled.

Amputation. 833 femurs were amputated and 218 (26 per cent) died. It is generally believed that the earlier the amputation the lower the mortality, but as yet it has been impossible to secure from the Surgeon-General's office sufficiently accurate data upon this question, in the majority of cases the time of operation was not recorded.

Splints. The Thomas splint was generally applied in the front lines and many fractured femurs received the additional benefit of suspension traction in the Balkan frame, for a time, in some one of the different hospitals through which they passed. Later, when ready for transportation to the United States, plaster casts were applied and many remained unchanged for a longer period than necessary, thus predisposing to joint stiffness. Consequently, the good or bad result obtained in many cases could not be attributed to any special method, as the fractures had been treated by so many various methods.

While in many cases the Thomas splint was not used efficiently, as it did not fit well, and the so-called extension straps were rarely found tight enough, nevertheless it is universally conceded to have proven by far the best method of treatment.

Suspension-traction, when accurately applied and efficiently maintained, produced really wonderful results, such as had not been generally believed possible before the World War, as Page, Pierson, Sinclair, Blake, Depage and Tuffier and others have demonstrated.

STATISTICS. From the 5063 fractured femurs, 158 (3.12 per cent) returned to duty in the army.

March 1, 1919, reports in the Surgeon-General's office indicated that a prognosis could be made that 76 per cent of fractures of long bones would recover with satisfactory function. How widely this has varied is seen by the follow-up reports of the Bureau of War Risk over two years later, May 1, 1921, when it was found that 70 per cent of fractures were suffering a disability of at least 20 per cent. Only 4 per cent have been reported, with a disability of less than 10 per cent.

Femurs. Of 2306 cases recorded in the Bureau of War Risk June 1, 1921, 83 per cent are suffering with a disability of 20 per cent or more; 0.02 per cent have less than 10 per cent disability.

Shortening is variable, but generally least when the Thomas or the Hodgen splint was well-applied and suspended from the Balkan frame. It was reported that there was no shortening in 18 per cent. I think this is doubtful, and more careful measurements

will show some shortening; 14 per cent had shortening of one-half to one inch; 72 per cent had shortening of one and a quarter inches or more.

Bowing. Outward bowing and marked deformity has occurred in many cases after union had been thought to be firm. This has occurred more often in the United States patients than among the British, who customarily wear a walking caliper splint for a considerable period.

Refracture has also occurred more frequently than is generally appreciated, and again more often with our patients than with the British. This accident, generally preventable, furnishes an additional reason for applying a walking caliper as soon as the patient leaves his bed to begin walking. Walking calipers have seldom been used by our patients.

Duration in Hospital. Five per cent of cases were discharged under three hundred days, 24 per cent under four hundred days, and 76 per cent required hospital treatment for more than four hundred days. This last figure varies considerably from that of three hundred and eight days as estimated from the records in the Surgeon-General's office—another illustration of the great difficulties in comparing records for the purpose of establishing standards for duration of disability.

During 1917, 1918 and 1919 there occurred in the United States Army, either in the United States or in the A. E. F., accidental fractures other than battle fractures.

Compound Fractures: 9449 cases required hospital treatment, with an average of sixty-nine days; 683 were femur fractures and required hospital treatment for an average of one hundred and five days.

Simple Fractures: 43,497 cases required an average of forty days' hospital treatment; 1531 were femur fractures, which required hospital treatment for an average of eighty-nine days.

Duration of Disability. When one consults the British fracture report to learn the period of disability for fractures of the femur he finds it is two hundred and thirty-one days; the American Fracture Committee gives two hundred and seventeen days;

the New York State Compensation gives one hundred and sixty-nine days; the United States Employers' Commission gives one hundred and fifty-two days; the United States Surgeon-General's Office gives eighty-nine days.

Compound Fractures of the Femur: Accidental, Surgeon-General's Office, one hundred and five days; battle injuries, three hundred and eight days; Bureau of War Risk, over four hundred days.

Non-union. Thus far there have been 906 cases reported: Humerus, 164; radius, 178; ulna, 120; radius and ulna, 71; femur, 155; tibia, 132; fibula, 9; tibia and fibula, 77. Bone grafts were used in 611 cases; Lane plates in 189; suture and wire in 52; kangaroo tendon or chromic gut in 54.

Grafts were taken from the tibia in 358 cases, and in 98 cases a sliding graft was employed; in 25 cases pegs made from beef bone were used, and in 31 cases pieces of rib.

Grafts: Autogenous, taken from the tibia, have proven the most efficient material for fractures of the long bones, on account of its characteristic strength. They are best made with the saw, and exact coaptation of the parts of the graft to respective parts of the host bone must be secured. Where many of these records are as yet too incomplete to give the final end-results, yet sufficient evidence has been secured to prove that bone-grafting is the most efficient method of treatment for non-union of fractures, and favorable results can be obtained in the largest percentage of cases.

Conclusions. The earliest possible reduction of a fracture is imperative. The suspension-traction method secures the best results in fractures of the long bones, and especially in the femur. It is easily adjusted; wounds of the thigh can be readily exposed for dressings; both the wounds and the fracture can be properly treated together without disturbing the satisfactory reduction of the fracture that is preserving the immovableness of the fragments. "It secures relaxation of the deforming muscles and so permits the bone fragments to fall into their proper relation." It permits mobilization of the joints of the part from the first

moment of beginning treatment and thus secures prompt recovery with preservation of function.

Fractures of the lower third with displacement are best treated by *skeletal* traction, with non-penetrating calipers above the knee, combined with early movement of the joints, hip, knee and ankle.

One of the most important lessons learned is the great *emphasis* placed upon *ultimate function* in the individual rather than on restoration of the contour and anatomical structure of a part. The former idea that marked deformity and disability are inevitable in fractures must be abandoned. In the past, surgeons have failed to devote the care which these most serious cases demanded. Putting into actual practice the methods which were demonstrated the best, especially in the last year of the World War, convalescence will be shortened, compensation reduced and the injured returned to his former occupation with a saving of 25 to 50 per cent of the time now lost—an enormous saving to industry.

Page and LeMesurier secured excellent results in 125 cases of fracture of the femur with an average shortening of 0.37 inch.

Pierson, in 68 cases, had no shortening in 29; 29 additional cases averaged 1.5 cm. shortening; mobility of the hip and the ankle were in almost all cases normal; in the knee, 55 had a range of flexion over 90 degree 10 had a range of between 60 degree and 90 degree and 3 ranged between 30 degree and 60 degree.

Bohler had 54 cases, in which the average shortening did not exceed one-half inch.

Erlacker and Zollinger reported equally good results.

Methods of treatment should be standardized and then universally employed. Conventional treatment is now inadequate. "There is no standard to which the advocate of efficiency may appeal; no incentive to energy, nor penalty for incompetence." In the past inefficiency has been tolerated; hereafter the surgeon who applies inadequate methods will be held responsible for failure. Jones states, "The question of fracture of the femur is essentially one of preventable surgery," dependent upon (1)

efficient fixation of the fracture with as correct alignment as possible, and (2) early and frequent movements of the neighboring joints.

RECOMMENDATIONS. The general adoption of the standard forms of splints used in the A. E. F.

General use of suspension-traction methods, employing the Hodgen or Thomas splints with the Balkan frame. Systematic and frequent *x*-ray examinations and mensuration to control the position of the fragments. Films should be taken as often as once every two weeks until firm union occurs. A portable *x*-ray outfit and a fracture table should be installed in every hospital where fractures are treated.

Physiotherapeutic massage and electrical treatment should be employed in every case at the earliest date.

Every femur case should wear a walking caliper when out of bed until union is solid.

Standard fracture records should be used in every case, and the greatest improvement in the treatment of fractures will result. Wherever these have been used there fractures have received the best treatment. The importance of using follow-up reports cannot be exaggerated.

Medical schools should give more clinical instruction in hospital wards in the actual detailed care of fractures.

One of the most valuable lessons learned from the World War was clearly demonstrated in the British and the United States A. E. F. hospitals, that where fractures were concentrated under one control in special services there the best results followed. It is firmly believed that civil hospitals should be so reorganized that fractures can be concentrated in special fracture services associated with a well-equipped physiotherapeutic department and that thereafter great improvement will result. Wherever this plan has been developed it has proven wise, as in the Massachusetts General, Montreal General and Bellevue hospitals.

According to Hoffa, two-thirds of all accident claims are based upon the consequence of fractures. Increased efforts must be made to so treat fractures as to obtain good functional

results in a larger number of cases. Economically it is the soundest business for the individual and for the state to get a perfect result in a young man. An imperfect cure makes a man face difficulties in finding employment and results in the permanent charge to the state of an unproductive, subsidized and discontented citizen.

SIMPLE FRACTURES OF THE HUMERUS[1]

By THOMAS W. HUNTINGTON, M.D.
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Table I shows that 38 per cent. of the cases of fractures of the humerus were treated by the operative method. This is probably an indication that anatomical reduction of these fractures without the assistance of vision directly upon the fragments is very difficult in a large number of cases.

Table III shows that 70 per cent. of the non-operative cases were followed by good anatomical results, and that when this was obtained 95 per cent. of the cases had good functional results and only a little over 1 per cent. had bad functional results. When only moderate anatomical results were obtained there followed barely 68 per cent. of good functional results.

This emphasizes the importance and value of good anatomical results in all cases of fracture of the humerus. (See Table IX.)

Table VIII indicates that fracture of the upper and lower extremities (the head and condyle) of the humerus required the greater number of operations, but even after operative treatment the anatomical results in these regions were not as good as by the non-operative treatment. The functional results were not nearly as good by the operative as by the non-operative treatment.

In Table V only a very small group could be obtained for the study of the influence of age upon the functional results. While the actual percentages would show the senile age period, viz., over sixty years, had better functional results than the 1 to 15 year

[1] The conclusions and data worked out by the Chairman.

period, the group is too small to disprove the general rule that children under fifteen years of age have better functional results after fractures than do those over fifteen years of age. The operative group in age periods is entirely too small for one to draw any definite conclusions.

In Table IX is shown that good anatomical results are followed in 79 per cent. of the cases by the shortest disability.

It is interesting to note that two cases, one an individual in the 1 to 15 age period and the other in the 45 to 60 period, notwithstanding a bad anatomical result, are rated among the cases of shortest disability. These anomalies are certainly exceptions, and apart from the fact that there are only two cases out of thirty-six, it is shown in Table X that the 45 to 60 age period cases had bad functional results and should doubtless be classed as permanently disabled.

In a group of thirty-six cases the average disability of the age period 1 to 15 years is three months, average of all age periods over 1 to 15 years is eight months, but of these 52 per cent. were reported to have a disability under three months.

Compound Fractures of the Humerus. In this group the number of cases is too small for any deductions. A little more than twice as many of the cases were treated by the operative as by the non-operative method. The tables suffice to show the various divisions of the study and the conclusions so far as they may be drawn from the small number of cases.

Simple Fractures of the Humerus

TABLE I

	Total.	Non-operative.	Operative.
Humerus, shoulder	42	26	16
Humerus, shaft	42	30	12
Humerus, elbow	78	61	17
	162	117	45

In the following tables the fraction percentages are not worked out:

TABLE II

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good . . .	116 (71%)	97 (83%)	15 (12%)	4 (3%)
Moderate . .	38 (23%)	21 (55%)	15 (39%)	2 (5%)
Bad	8 (4%)	3 (37%)	2 (25%)	3 (37%)
	162 (98%)	121 (74%)	32 (19%)	9 (5%)

TABLE III.—NON-OPERATIVE

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good . . .	82 (70%)	78 (95%)	3 (3%)	1 (1%)
Moderate . .	29 (24%)	20 (68%)	8 (27%)	1 (3%)
Bad	6 (5%)	3 (50%)	2 (33%)	1 (16%)
	117 (99%)	101 (86%)	13 (11%)	3 (2%)

TABLE IV.—OPERATIVE

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good . . .	34 (75%)	19 (55%)	12 (35%)	3 (6%)
Moderate . .	9 (20%)	1 (11%)	7 (77%)	1 (11%)
Bad	2 (4%)	0	0	2 (100%)
	45 (99%)	20 (44%)	19 (42%)	6 (13%)

TABLE V.—INFLUENCE OF AGE ON THE FUNCTIONAL RESULTS

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good . .	8 (72%)	7 (87%)	1 (12%)	0
Moderate .	2 (18%)	1 (50%)	0	1 (50%)
Bad . . .	1 (9%)	1 (100%)	0	0
	11 (99%)	9 (81%)	1 (9%)	1 (9%)
15 to 45 years:				
Good . . .	10 (90%)	7 (70%)	3 (30%)	0
Moderate .	1 (9%)	0	1 (100%)	0
Bad . . .	0 (0%)	0	0	0
	11 (99%)	7 (63%)	4 (36%)	0
45 to 60 years:				
Good . .	6 (75%)	4 (66%)	2 (33%)	0
Moderate .	1 (12%)	1 (100%)	0	0
Bad . . .	1 (12%)	1 (100%)	0	0
	8 (99%)	6 (75%)	2 (25%)	0

Over 60 years:				
Good . . .	5 (83%)	5 (100%)	0	0
Moderate .	1 (16%)	0	1 (100%)	0
Bad . . .	0	0	0	0
	6 (99%)	5 (83%)	1 (16%)	0
All over 15 years:				
Good . . .	21 (84%)	16 (76%)	5 (23%)	0
Moderate .	3 (8%)	1 (33%)	2 (66%)	0
Bad . . .	1 (4%)	0	0	1 (100%)
	25 (96%)	17 (68%)	7 (28%)	1 (4%)

TABLE VI.—INFLUENCE OF AGE ON NON-OPERATIVE TREATMENT

Anatomical result.	Total.	Good.	Functional result. Moderate.	Bad.
1 to 15 years:				
Good . . .	7 (70%)	7 (100%)	0	0
Moderate .	2 (20%)	2 (100%)	0	0
Bad . . .	1 (10%)	1 (100%)	0	0
	10 (100%)	10 (100%)	0	0
15 to 45 years:				
Good . .	7 (87%)	6 (85%)	1 (14%)	0
Moderate .	1 (12%)	0	1 (100%)	0
Bad . . .	0	0	0	0
	8 (99%)	6 (75%)	2 (25%)	0
45 to 60 years:				
Good . . .	4 (80%)	2 (50%)	2 (50%)	0
Moderate .	1 (20%)	1 (100%)	0	0
Bad . . .	0	0	0	0
	5 (100%)	3 (60%)	2 (40%)	0
Over 60 years:				
Good . . .	2 (66%)	2 (100%)	0	0
Moderate .	1 (33%)	0	1 (100%)	0
Bad . . .	0	0	0	0
	3 (99%)	2 (66%)	1 (33%)	0
All over 15 years:				
Good . . .	13 (81%)	10 (76%)	3 (23%)	0
Moderate .	3 (18%)	1 (33%)	2 (66%)	0
Bad . . .	0	0	0	0
	16 (99%)	11 (68%)	5 (31%)	0

TABLE VII.—INFLUENCE OF AGE ON OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good . . .	1 (100%)	0	1 (100%)	0
Moderate .	0	0	0	0
Bad . . .	0	0	0	0
	1 (100%)	0	1 (100%)	0
15 to 45 years:				
Good . . .	3 (100%)	2 (66%)	1 (33%)	0
Moderate .	0	0	0	0
Bad . . .	0	0	0	0
	3 (100%)	2 (66%)	1 (33%)	0
45 to 60 years:				
Good . . .	2 (100%)	2 (100%)	0	0
Moderate .	0	0	0	0
Bad . . .	0	0	0	0
	2 (100%)	2 (100%)	0	0
Over 60 years:				
Good . .	3 (100%)	2 (66%)	1 (33%)	0
Moderate .	1 (25%)	0	1 (100%)	0
Bad . . .	0	0	0	0
	4 (100%)	2 (50%)	2 (50%)	0
All over 15 years:				
Good . . .	8 (88%)	6 (75%)	2 (25%)	0
Moderate .	1 (11%)	0	1 (100%)	0
Bad . . .	0	0	0	0
	9 (99%)	6 (66%)	3 (33%)	0

TABLE VIII

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		NON-OPERATIVE		
Shoulder:				
Good . . .	17 (65%)	16 (94%)	0	1 (5%)
Moderate .	6 (23%)	4 (66%)	2 (33%)	0
Bad . . .	3 (11%)	1 (33%)	2 (66%)	0
	26 (99%)	21 (81%)	4 (15%)	1 (3%)
		OPERATIVE		
Shoulder:				
Good . . .	10 (62%)	7 (70%)	2 (20%)	1 (10%)
Moderate .	5 (31%)	1 (20%)	4 (80%)	0
Bad . . .	1 (6%)	0	0	1 (100%)
	16 (99%)	8 (50%)	6 (37%)	2 (12%)

Non-operative				
Shaft:				
Good . . .	18 (60%)	15 (83%)	3 (16%)	0
Moderate .	11 (36%)	7 (63%)	3 (27%)	1 (9%)
Bad . . .	1 (3%)	1 (100%)	0	0
	30 (99%)	23 (76%)	6 (20%)	1 (3%)
Operative				
Shaft:				
Good . . .	10 (83%)	4 (40%)	5 (50%)	1 (10%)
Moderate .	1 (8%)	0	1 (100%)	0
Bad . . .	1 (8%)	0	0	1 (100%)
	12 (99%)	4 (33%)	6 (50%)	2 (16%)
Non-operative				
Elbow:				
Good . . .	47 (77%)	47 (100%)	0	0
Moderate .	12 (19%)	9 (75%)	3 (25%)	0
Bad . . .	2 (3%)	1 (50%)	1 (50%)	0
	61 (99%)	57 (93%)	4 (6%)	0
Operative				
Elbow:				
Good . .	14 (82%)	8 (57%)	5 (35%)	1 (7%)
Moderate .	3 (17%)	0	2 (66%)	1 (33%)
Bad . . .	0	0	0	0
	17 (99%)	8 (47%)	7 (41%)	2 (11%)

TABLE IX.—DOES GOOD ANATOMICAL RESULTS LESSEN PERIOD OF DISABILITY?

Anatomical result.	Total.	Period of disability.				
		Under 3 months.	Under 6 months.	Under 9 months.	Under 12 months.	Over 12 months.
Humerus, all regions:						
Good . .	29 (81%)	23 (79%)	6 (20%)	0	0	0
Moderate .	5 (13%)	1 (20%)	4 (80%)	0	0	0
Bad . . .	2 (5%)	2 (100%)	0 (0%)	0	0	0
Total . . .	36 (100%)	26 (72%)	10 (27%)	0	0	0

TABLE X.—HUMERUS, ALL FORMS—36 CASES

	Anatomical result.	Total.	Period of disability and functional result.											
			Under 3 months.			Under 6 months.			Under 9 months.			Under 12 months.		
			Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.
1 to 15 years	Good	8	7	0	0	0	1	0	0	0	0	0	0	0
	Moderate	2	1	0	0	1	0	0	0	0	0	0	0	0
	Bad	1	1	0	0	0	0	0	0	0	0	0	0	0
	Total	11	9	0	0	1	1	0	0	0	0	0	0	0
15 to 45 years	Good	10	5	1	0	2	0	0	0	1	0	0	1	0
	Moderate	1	0	0	0	0	1	0	0	0	0	0	0	0
	Bad	0	0	0	0	0	0	0	0	0	0	0	0	0
	Total	11	5	1	0	2	1	0	0	1	0	0	1	0
45 to 60 years	Good	6	3	0	0	1	1	0	0	1	0	0	0	0
	Moderate	1	0	0	0	0	0	0	1	0	0	0	0	0
	Bad	1	0	0	1	0	0	0	0	0	0	0	0	0
	Total	8	3	0	1	1	1	0	1	1	0	0	0	0
Over 60 years	Good	5	3	0	0	1	1	0	0	0	0	0	0	0
	Moderate	1	0	0	0	0	1	0	0	0	0	0	0	0
	Bad	0	0	0	0	0	0	0	0	0	0	0	0	0
	Total	6	3	0	0	1	2	0	0	0	0	0	0	0
All ages	Good	29	18	1	0	4	3	0	0	2	0	0	1	0
	Moderate	5	1	0	0	1	2	0	1	0	0	0	0	0
	Bad	2	1	0	1	0	0	0	0	0	0	0	0	0
	Total	36	20	1	1	5	5	0	1	2	0	0	1	0

Average disability for all ages, 5.33 months.

TABLE XI.—OPERATIVE COMPARED WITH NON-OPERATIVE TREATMENT (HUMERUS)

	Total.	Period of disability.				
		Under 3 months.	Under 6 months.	Under 9 months.	Under 12 months.	Over 12 months.
1 to 15 years:						
Non-operative .	10 (90%)	9 (90%)	1 (10%)	0	0	0
Operative . .	1	0	1 (100%)	0	0	0
15 to 45 years:						
Non-operative .	8 (72%)	5 (62%)	2 (25%)	1 (12%)	0	0
Operative . .	3 (27%)	2 (66%)	0	0	0	0
45 to 60 years:						
Non-operative .	6 (75%)	3 (50%)	2 (33%)	1 (16%)	0	0
Operative . .	2 (25%)	1 (50%)	1 (50%)	0	0	0
Over 60 years:						
Non-operative .	3 (50%)	1 (33%)	2 (66%)	0	0	0
Operative . .	3 (50%)	2 (66%)	1 (33%)	0	0	0
All ages:						
Non-operative .	27 (75%)	18 (66%)	7 (25%)	2 (7%)	0	0
Operative . .	9 (25%)	5 (55%)	3 (33%)	0	1 (11%)	0

The series is too small to draw absolute conclusions. It appears from the above, however, that operative treatment lengthens the period of disability.

TABLE XII.—INFLUENCE ON RESULT OF TIME ELAPSING BETWEEN INJURY AND OPERATION (HUMERUS)

No. of cases.		Ages, years.		Date of operation, days.					Anatomical result.	Functional result.		
1 to 15	15 to 45	45 to 60	Over 60	0 to 7	8 to 14	15 to 21	22 to 28	29		Good.	Mod.	Bad.
1	0	0	0	0	1	0	0	0	Good . . 1	0	1	0
									Moderate . 0	0	0	0
									Bad . . . 0	0	0	0
	3	0	0	1	0	1	0	1	Good . . 3	1	2	0
									Moderate . 0	0	0	0
									Bad . . . 0	0	0	0
		2	0	1	0	1	0	0	Good . . 2	2	0	0
									Moderate . 0	0	0	0
									Bad . . . 0	0	0	0
			3	1	1	1	0	0	Good . . 3	2	1	0
									Moderate . 0	0	0	0
									Bad . . . 0	0	0	0
	6	0	0	3	2	3	1	1	Good . . 9	5	4	0
									Moderate . 0	0	0	0
									Bad . . . 0	0	0	0
										5	4	0

Compound Fractures of the Humerus

TABLE I

	Total.	Non-operative.	Operative.
Humerus, shoulder	4	2	2
Humerus, shaft	15	4	11
Humerus, elbow	0	0	0
	19	6	13

TABLE II

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	12 (63%)	11 (91%)	1 (8%)	0
Moderate	4 (21%)	1 (25%)	3 (75%)	0
Bad	3 (15%)	0	0	3 (100%)
	19 (99%)	12 (63%)	4 (21%)	3 (15%)

TABLE III.—NON-OPERATIVE

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	6 (100%)	6 (100%)	0	0
Moderate	0	0	0	0
Bad	0	0	0	0
	6 (100%)	6 (100%)	0	0

TABLE IV.—OPERATIVE

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	6 (46%)	5 (83%)	1 (16%)	0
Moderate	4 (30%)	1 (25%)	3 (75%)	0
Bad	3 (23%)	0	0	3 (100%)
	13 (99%)	6 (46%)	4 (30%)	3 (23%)

TABLE V.—INFLUENCE OF AGE ON THE FUNCTIONAL RESULT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good	3 (75%)	3 (100%)	0	
Moderate	0	0	0	
Bad	1 (25%)	0	0	1 (100%)
	4 (100%)	3 (75%)	0	1 (25%)

15 to 45 years:				
Good	8 (57%)	6 (75%)	2 (25%)	0
Moderate	4 (28%)	1 (25%)	3 (75%)	0
Bad	2 (14%)	0	0	2 (100%)
	14 (99%)	7 (50%)	5 (35%)	2 (14%)
45 to 60 years:				
Good	1 (100%)	1 (100%)	0	0
Moderate	0	0	0	0
Bad	0	0	0	0
	1 (100%)	1 (100%)	0	0
All over 60 years, no cases.				
All over 15 years:				
Good	9 (60%)	7 (77%)	2 (22%)	0
Moderate	4 (25%)	1 (25%)	3 (75%)	0
Bad	2 (13%)	0	0	2 (100%)
	15 (98%)	8 (53%)	5 (33%)	2 (13%)

TABLE VI.—INFLUENCE OF AGE ON THE FUNCTIONAL RESULTS (NON-OPERATIVE TREATMENT)

Anatomical result.	Total.	Good.	Functional result. Moderate.	Bad.
1 to 15 years:				
Good	3 (100%)	3 (100%)	0	0
Moderate	0	0	0	0
Bad	0	0	0	0
	3 (100%)	3 (100%)	0	0
15 to 45 years:				
Good	1 (100%)	1 (100%)	0	0
Moderate	0	0	0	0
Bad	0	0	0	0
	1 (100%)	1 (100%)	0	0
45 to 60 years:				
Good	2 (100%)	2 (100%)	0	0
Moderate	0	0	0	0
Bad	0	0	0	0
	2 (100%)	2 (100%)	0	0
All over 60 years, no cases.				
All over 15 years:				
Good	3 (100%)	3 (100%)	0	0
Moderate	0	0	0	0
Bad	0	0	0	0
	3 (100%)	3 (100%)	0	0

TABLE VII.—INFLUENCE OF AGE ON OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good . . .	0	0	0	0
Moderate .	0	0	0	0
Bad . . .	1 (100%)	0	0	1 (100%)
	1 (100%)	0	0	1 (100%)
15 to 45 years:				
Good . .	7 (58%)	5 (71%)	2 (28%)	0
Moderate .	3 (25%)	1 (33%)	2 (66%)	0
Bad . . .	2 (16%)	0	0	2 (100%)
	12 (99%)	6 (50%)	4 (33%)	2 (16%)

45 to 60 years, no cases. Over 60 years, no cases.

TABLE VIII

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		NON-OPERATIVE		
Shoulder:				
Good . .	2 (50%)	2 (100%)	0	0
Moderate .	0	0	0	0
Bad . . .	0	0	0	0
	2 (100%)	2 (100%)	0	0
		OPERATIVE		
Shoulder:				
Good . .	0	0	0	0
Moderate .	0	0	0	0
Bad . . .	2 (100%)	0	0	2
	2 (100%)	0	0	2 (100%)
		NON-OPERATIVE		
Shaft:				
Good . .	3 (100%)	3 (100%)	0	0
Moderate .	0	0 (100%)	0	0
Bad . . .	0	0	0	0
	3 (100%)	3 (100%)	0	0

		Operative		
Shaft:				
Good . . .	7 (58%)	5 (71%)	2 (28%)	0
Moderate .	4 (33%)	1 (25%)	3 (75%)	0
Bad . . .	1 (8%)	0	0	1 (100%)
	12 (99%)	6 (50%)	5 (41%)	1 (8%)

Elbow, non-operative, no cases.
Elbow, operative, no cases.

TABLE IX.—COMPARATIVE RESULTS OF OPERATIVE AND NON-OPERATIVE CASES OF COMPOUND FRACTURES OF THE HUMERUS

	Operative cases.			Non-operative cases.		
	Cases.	Good anatomical result.	Good functional result.	Cases.	Good anatomical result.	Good functional result.
Humerus, shoulder .	2	0	0	2	100%	100%
Humerus, shaft .	3	100%	100%	12	58%	71%
Humerus, elbow .	0	0	0	0	0	0

FRACTURES OF THE BONES OF THE FOREARM

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(By Invitation.)

The non-operative treatment of fractures of the shafts of the radius and ulna gave 83 per cent. good functional, 77 per cent. good anatomical and 1 per cent. bad results. Operative methods gave 83 per cent. good functional results, 50 per cent. good anatomical results, 17 per cent. bad functional results. (Table VII.)

Non-operative treatment of fractures near joints gave 79 per cent. good functional and 77 per cent. good anatomical results. Operative treatment gave 72 per cent. good functional and 27 per cent. good anatomical results. (Table VIII.)

Disability was not longer than six weeks in 45 per cent. of the 257 cases and in 57 per cent. of the cases with good anatomical results. (Table X.)

The non-operative treatment of compound fractures gave 55 per cent. good functional results and the operative treatment gave 14 per cent. good functional results. The number of cases, 16, is too small for conclusions of value. (Table XI.)

In so far as conclusions are justified by a study and classification of the reported cases of fracture of the forearm bones it would seem that:

Non-operative treatment gave good functional results in 78 per cent. as compared to 68 per cent. operative, and showed only 2 per cent. bad functional results as compared to 13 per cent. bad functional results in the operative cases.

The cases operated on were in the main those in which injury was most extensive and delay in adequate treatment most pronounced. (Table III.)

Forearm fractures in the young (under fifteen years) gave the best results, both under conservative and operative treatment, if one small series (6 cases over sixty years of age) be excluded. (Tables IV, V, VI.)

Internal Fixation. Of the 43 cases with data only 36 are recorded with sufficient detail to be of service. Bone plates were used in 17 cases; catgut or tendon in 7; screws in 5; bone inlays in 4; and wire in 2. In 5 cases in the series the internal fixation had to be removed later (three bone inlays and two bone plates). In 3 of the 5 cases, 7.9 per cent. of the total number, infection seemed to be the cause.

Time of Operation. The earliest operation was within three hours; the latest was one hundred and twenty days. Only 4 cases were operated under forty-eight hours. Table XII shows that the best anatomical and functional results are obtained under early operation; that is, in the first and second weeks, each giving 50 per cent. good results. After the fourth week the results are over 50 per cent. bad for function.

TABLE I

	Total.	Non-operative.	Operative.
Radius, head	6	3	3
Radius, shaft	41	38	3
Radius, Colles's	100	95	5
Ulna, olecranon	15	12	3
Ulna, shaft	32	28	4
Radius and ulna, shafts	76	50	26
Total	270	226	44

Note.—In reading the following tables there will be found some discrepancies which, however, are due to incomplete history records and insufficient data. The percentage figures are always taken as the first whole number and no fractions considered.

TABLE II.—FRACTURES OF THE RADIUS AND ULNA

Anatomical result.		Functional result.							
		Total.		Good.		Moderate.		Bad.	
		Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.
Radius, Shaft	Good	33	2	33	2	0	0	0	0
	Moderate	5	0	2	0	3	0	0	0
	Bad	0	0	0	0	0	0	0	0
	Total	38	2	35	2	3	0	0	0
	Per cent.	100	100	90	100	70	0	0	0
Radius, Colles's	Good	70	2	65	2	5	0	0	0
	Moderate	13	2	4	1	7	1	2	0
	Bad	10	1	3	0	6	..	1	1
	Total	93	5	72	3	18	1	3	1
	Per cent.	100	100	77	60	0	20	60	20
Ulna, Shaft	Good	20	2	15	2	5	0	0	0
	Moderate	5	..	3	..	2	0	0	0
	Bad	3	2	2	..	1	1	0	1
	Total	28	4	20	2	8	1	0	1
	Per cent.	100	100	71	50	28	25	0	25
Radius and Ulna, Shafts	Good	26	12	24	10	2	2	0	0
	Moderate	13	8	6	2	7	6	0	0
	Bad	7	6	0	1	5	1	2	4
	Total	46	26	30	13	14	9	2	4
	Per cent	100	100	65	50	30	34	4	15
Radius, Head	Good	3	1	3	1	0	0	0	0
	Moderate	0	2	0	1	0	1	0	0
	Bad	0	0	0	0	0	0	0	0
	Total	3	3	3	2	0	1	0	0
	Per cent	100	100	100	66	0	33	0	0
Ulna, Olecranon	Good	10	0	10	0	0	0	0	0
	Moderate	1	3	0	3	1	0	0	0
	Bad	0	0	0	0	0	0	0	0
	Total	11	3	10	3	1	0	0	0
	Per cent	100	100	90	100	9	0	0	0
	Grand total	219	43	170	25	44	12	5	6
	Per cent.	100	100	78	68	20	27	2	13

TABLE III.—FRACTURES OF THE RADIUS AND ULNA.

Anatomical result.	Functional result.							
	Total.		Good.		Moderate.		Bad	
	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.
Good . . .	162	19	150	17	12	2	0	0
Moderate . .	37	15	15	7	20	8	2	0
Bad . . .	20	9	5	1	12	2	3	6
Total . .	219	43	170	25	44	12	5	6
Per cent. . .	100	100	78	68	20	27	2	13

NOTE.—Only 4 cases were operated under forty-eight hours; 2 had good functional and anatomical results; 2 had bad functional and anatomical results.

TABLE IV.—INFLUENCE OF AGE ON THE FUNCTIONAL RESULT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Under 15 years:				
Good . . .	78	74	3	1
Moderate . .	18	13	5	0
Bad	8	3	4	1
Total . . .	104	90 (86%)	12 (11%)	2 (19%)
15 to 45 years:				
Good . . .	91	85	4	2
Moderate . .	12	3	8	1
Bad	11	2	4	5
Total . . .	114	90 (78%)	16 (14%)	8 (7%)
45 to 60 years:				
Good . . .	15	12	3	0
Moderate . .	8	5	3	0
Bad	7	1	3	3
Total . . .	30	18 (60%)	9 (30%)	3 (10%)
Over 60 years:				
Good . . .	7	6	1	0
Moderate . .	6	0	6	0
Bad	3	1	2	0
Total . . .	16	7 (43%)	9 (56%)	0
All over 15 years:				
Good . . .	113	103	9	2
Moderate . .	26	8	7	4
Bad	21	4	9	8
Total . . .	160	115 (72%)	25 (15%)	14 (8%)

TABLE V.—INFLUENCE OF AGE ON NON-OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Under 15 years:				
Good	68	64	3	1
Moderate	16	13	3	0
Bad	4	2	1	1
Total	88	79 (89%)	7 (7%)	2 (2%)
15 to 45 years:				
Good	81	77	4	0
Moderate	7	1	5	1
Bad	7	2	4	1
Total	95	80 (84%)	13 (13%)	2 (2%)
45 to 60 years:				
Good	15	12	3	0
Moderate	8	5	3	0
Bad	5	1	3	1
Total	28	18 (64%)	9 (62%)	1 (3%)
Over 60 years:				
Good	6	6	0	0
Moderate	6	0	6	0
Bad	3	1	2	0
Total	15	7 (46%)	8 (52%)	0
All over 15 years:				
Good	102	95	8	0
Moderate	22	6	4	1
Bad	15	4	9	2
Total	139	105 (75%)	21 (15%)	3 (2%)

TABLE VI.—INFLUENCE OF AGE ON OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Under 15 years:				
Good	10	10	0	0
Moderate	2	0	2	0
Bad	4	1	2	0
Total	16	11 (68%)	4 (25%)	0

15 to 45 years:				
Good	10	8	0	2
Moderate	5	2	3	0
Bad	4	0	0	4
Total	19	10 (52%)	3 (15%)	6 (31%)
45 to 60 years:				
Good	0	0	0	0
Moderate	0	0	0	0
Bad	2	0	0	2
Total	2	0	0	2 (100%)
Above 60 years:				
Good	1	0	1	0
Moderate	0	0	0	0
Bad	0	0	0	0
Total	1	0	1 (100%)	0
All over 15 years:				
Good	11	8	1	2
Moderate	5	2	3	0
Bad	6	0	0	6
Total	22	10 (45%)	4 (18%)	8 (37%)

TABLE VII.—FRACTURES OF SHAFTS OF THE LONG BONES

NON-OPERATIVE

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	79	72	7	0
Moderate	23	11	12	0
Bad	10	2	6	2
Total	112	85 (83%)	25 (24%)	2 (1%)

This series does not contain Colles's, olecranon or radial head fractures.

OPERATIVE

Good	16	14	2	0
Moderate	8	2	6	0
Bad	8	1	2	5
Total	32	17 (53%)	10 (31%)	5 (17%)

TABLE VIII.—FRACTURES NEAR JOINTS

Non-operative

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	83	78	5	0
Moderate	14	4	8	2
Bad	10	3	6	1
Total	107	85 (79%)	19 (17%)	3 (2%)

Operative

Good	3	3	0	0
Moderate	7	5	2	0
Bad	1	0	0	1
Total	11	8 (72%)	2 (18%)	1 (9%)

TABLE IX

Good Functional Results Following Non-operative Treatment

Site.	Total.	Good function.
Radius and ulna, shafts	112	85
Radius and ulna, extremities	107	85
Total	219	170 (78%)

Good Functional Results Following Operative Treatment

Radius and ulna, shafts	32	17
Radius and ulna, extremities	11	8
Total	43	25 (68%)

TABLE X.—PERIOD OF DISABILITY IN FRACTURES OF THE UPPER EXTREMITY

Anatomical result.	Total.	Under 6 weeks.	Under 12 weeks.	Under 24 weeks.	Over 24 weeks.
Good	174	100	55	12	7
Moderate	50	16	23	6	5
Bad	33	12	10	6	5
Total	257	128 (45%)	88 (33%)	24 (9%)	17 (8%)

NOTE.—This table shows that good anatomical results were associated with disability under six weeks in 45 per cent. of cases. Disability here had been taken to apply to time elapsing before return to work.

TABLE XI.—COMPOUND FRACTURE OF THE FOREARM GROUPED IN ONE SERIES BECAUSE OF SMALL NUMBER

Anatomical result.	Functional result.								Died.
	Total.		Good.		Moderate.		Bad.		
	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.	
Good	4	1	4	1	0	0	0	0	0
Moderate	2	3	0	0	2	3	0	0	0
Bad	3	3	1	0	0	1	3	1	2
Total	9	7	5	1	2	4	3	1	2
Per cent	0	0	53	14	22	57	33	14	0

Deaths: 1 from tetanus; 1 from shock and other injuries. Other deaths in the records were not attributable to the forearm condition.

TABLE XII.—TIME OF OPERATION ON FRACTURED FOREARM

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 7 days:				
Good	7	6	1	0
Moderate	3	0	3	0
Bad	2	0	0	2
Total	12 (100%)	6 (50%)	4 (33%)	2 (16%)
7 to 14 days:				
Good	5	4	1	0
Moderate	2	0	2	0
Bad	1	0	0	1
Total	8 (100%)	4 (50%)	3 (37%)	1 (12%)

14 to 21 days:				
Good	1	1	0	0
Moderate	3	0	3	0
Bad	1	0	0	1
Total	5 (100%)	1 (20%)	3 (60%)	1 (20%)
21 to 28 days:				
Good	No cases reported.			
Moderate				
Bad				
Total				
Over 28 days:				
Good	3	3	0	0
Moderate	1	0	1	0
Bad	5	0	0	5
Total	9 (100%)	3 (33%)	1 (11%)	5 (55%)

FRACTURES OF THE TIBIA AND FIBULA

By ASTLEY P. C. ASHHURST, M.D.
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The statistics presented herewith are based on the figures given in the report of this committee for the year 1915 with the addition of the cases since collected, making a total of 926 cases of fractures of the tibia and fibula which have been reported in sufficient detail to permit of study.

These cases are thus divided.

Simple fractures, 625 (non-operative, 555; operative, 70).

Compound fractures, 301 (non-operative, 184; operative, 117).

Total, 926 (non-operative, 739; operative, 187).

The thanks of the committee are extended to their collaborators, Dr. Otto C. Gaub, of Pittsburgh; Dr. Daniel F. Jones, of Boston; Dr. Kellogg Speed, of Chicago, as well as to Dr. Edward B. Hodge, of Philadelphia, for reports of cases; and especially to Dr. Irvine M. Boykin and Dr. Edward T. Crossan for collecting a very large number of end-results from the Episcopal Hospital of Philadelphia; also to Miss M. C. Lamor for tabulating the cases of fractures of the tibia and fibula.

The results are slightly better, in the more recently collected cases, than they were in those forming the subject of investigation in 1915; but in these latter, as well as in the earlier cases, these results are not ideal. They show that the treatment of fractures of the leg bones is still capable of much improvement.

The accompanying tables analyze separately all the simple fractures (Tables I to XIII inclusive) and subsequently (Tables XIV to XXI inclusive) the compound fractures.

Simple Fractures of the Leg Bones. These statistics (Tables I to XIII inclusive) relate only to recent, closed fractures.

Included in the analysis are the following 625 individual cases of fracture:

Simple Fractures of the Leg Bones, 1915 and 1921

TABLE I

	Total.	Non-operative.	Operative.
Fibula, shaft	46	46	
Tibia, shaft	114	92	22
Tibia and fibula, shafts	232	192	40
Tibia and fibula, ankle	109	101	8
Tibia, ankle	36	36	
Fibula, ankle	88	88	
	625	555	70

TABLE II

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	476 (76%)	408 (86%)	60 (12%)	8 (2%)
Moderate	109 (18%)	57 (51%)	40 (39%)	12 (10%)
Bad	40 (6%)	3 (8%)	14 (35%)	23 (57%)
	625 (100%)	468 (75%)	114 (18%)	43 (7%)

TABLE III.—NON-OPERATIVE

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	428 (77%)	372 (87%)	49 (11%)	7 (2%)
Moderate	94 (17%)	49 (52%)	36 (38%)	9 (10%)
Bad	33 (6%)	1 (3%)	12 (36%)	20 (61%)
	555 (100%)	422 (76%)	97 (17%)	36 (7%)

TABLE IV.—OPERATIVE

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	48 (69%)	36 (75%)	11 (23%)	1 (2%)
Moderate	15 (21%)	8 (53%)	4 (27%)	3 (20%)
Bad	7 (10%)	2 (28%)	2 (28%)	3 (44%)
	70 (100%)	46 (66%)	17 (24%)	7 (10%)

As in our previous report a much larger number of case records was examined, but many had to be rejected partly or entirely because of lack of data. This insufficiency in regard to certain details accounts for the fewer number of cases analyzed in some tables than in others.

We have tried to maintain the standard as set forth under the heading of *Definitions* on page 12 of our 1915 report. But in many of the reports received the examining surgeons appear to

TABLE V.—INFLUENCE OF AGE ON THE FUNCTIONAL RESULT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Under 15 years:				
Good	51 (88%)	49 (96%)	1 (2%)	1 (2%)
Moderate	6 (10%)	3 (50%)	3 (50%)	
Bad	1 (2%)	...	1 (100%)	
	58 (100%)	52 (89%)	5 (9%)	1 (2%)
15 to 45 years:				
Good	244 (80%)	217 (89%)	25 (10%)	2 (1%)
Moderate	48 (16%)	28 (58%)	18 (38%)	2 (4%)
Bad	11 (4%)	2 (18%)	6 (55%)	3 (27%)
	303 (100%)	247 (82%)	49 (16%)	7 (2%)
45 to 60 years:				
Good	70 (67%)	56 (80%)	11 (16%)	3 (4%)
Moderate	23 (22%)	11 (48%)	8 (35%)	4 (17%)
Bad	12 (11%)	...	5 (42%)	7 (58%)
	105 (100%)	67 (64%)	24 (23%)	14 (13%)
Over 60 years:				
Good	17 (77%)	12 (71%)	4 (23%)	1 (6%)
Moderate	3 (14%)	1 (33.3%)	1 (33.3%)	1 (33.4%)
Bad	2 (9%)	...	....	2 (100%)
	22 (100%)	13 (59%)	5 (23%)	4 (18%)
All over 15 years:				
Good	331 (77%)	285 (86%)	40 (12%)	6 (2%)
Moderate	74 (17%)	40 (55%)	27 (36%)	7 (9%)
Bad	25 (6%)	2 (8%)	11 (44%)	12 (48%)
	430 (100%)	327 (76%)	78 (18%)	25 (6%)

have been less strict than the committee, and therefore the statistical tables probably show better results than actually are obtained in practice. For instance, some reports received have classed as "good" anatomical results cases in which shortening of as much as 3 cm. was recorded; and as "good" functional results cases in which pain in damp weather persisted or where there was evident, but not disabling, limitation of motion and

TABLE VI.—INFLUENCE OF AGE ON NON-OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Under 15 years:				
Good . .	47 (90%)	46 (98%)	1 (2%)	
Moderate .	4 (8%)	2 (50%)	2 (50%)	
Bad . . .	1 (2%)	...	1 (100%)	
	52 (100%)	48 (92%)	4 (8%)	
15 to 45 years:				
Good . .	218 (81%)	199 (91%)	17 (8%)	2 (1%)
Moderate .	41 (16%)	23 (57%)	17 (41%)	1 (2%)
Bad . . .	9 (3%)	1 (11%)	5 (56%)	3 (33%)
	268 (100%)	223 (83%)	39 (15%)	6 (2%)
45 to 60 years:				
Good . .	66 (70%)	54 (81%)	9 (14%)	3 (5%)
Moderate .	20 (21%)	9 (45%)	8 (40%)	3 (15%)
Bad . . .	9 (9%)	...	5 (56%)	4 (44%)
	95 (100%)	63 (66%)	22 (23%)	10 (11%)
Over 60 years:				
Good . .	17 (77%)	12 (70%)	4 (24%)	1 (6%)
Moderate .	3 (14%)	1 (33.3%)	1 (33.3%)	1 (33.4%)
Bad . . .	2 (9%)	...	....	2 (100%)
	22 (100%)	13 (59%)	5 (23%)	4 (18%)
All over 15 years:				
Good . .	301 (78%)	267 (88%)	30 (10%)	6 (2%)
Moderate .	64 (17%)	33 (50%)	27 (42%)	5 (8%)
Bad . . .	20 (5%)	1 (5%)	10 (50%)	9 (45%)
	385 (100%)	299 (78%)	66 (17%)	20 (5%)

where disability was "partial," not entirely "absent." Our definitions showed that such results should have been classed as "moderate" or "bad" in anatomy and function according to the degree of deformity and disability; and though an effort was made to correct very glaring inaccuracies of this kind in the reports received, it is probable a number have been passed undetected.

Table II shows the functional result in all the cases reported; and it certainly is not creditable that only 75 per cent of the whole number of patients recovered good function. Table III

TABLE VII.—INFLUENCE OF AGE ON OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Under 15 years:				
Good . .	4 (67%)	3 (75%)	....	1 (25%)
Moderate .	2 (33%)	1 (100%)	1 (100%)	
Bad . . .				
	6 (100%)	4 (66%)	1 (17%)	1 (17%)
15 to 45 years:				
Good . .	26 (74%)	18 (69%)	8 (31%)	
Moderate .	7 (20%)	5 (72%)	1 (14%)	1 (14%)
Bad . . .	2 (6%)	1 (50%)	1 (50%)	
	35 (100%)	24 (68%)	10 (29%)	1 (3%)
45 to 60 years:				
Good . .	4 (40%)	2 (50%)	2 (50%)	
Moderate .	3 (30%)	2 (67%)	....	1 (33%)
Bad . . .	3 (30%)	...	....	3 (100%)
	10 (100%)	4 (40%)	2 (20%)	4 (40%)
Over 60 years:				
Good	....	....	....	....
Moderate	....	....	....	....
Bad . . .	....	....	....	....
All over 15 years:				
Good . .	30 (67%)	20 (67%)	10 (33%)	
Moderate .	10 (22%)	7 (70%)	1 (10%)	2 (20%)
Bad . . .	5 (11%)	1 (20%)	1 (20%)	3 (60%)
	45 (100%)	27 (62%)	12 (27%)	5 (11%)

and Table IV show the functional results in non-operative and operative cases respectively. It is especially worthy of note that in each case the greatest number of patients who secure good function are those in whom the anatomical result is good (87

TABLE VIII.—FRACTURES OF THE TIBIA AND THE FIBULA

Anatomical results.	Functional results.							
	Total.		Good.		Moderate.		Bad.	
	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.
SHAFTS (also Shaft of the Tibia alone)								
Good	206 (70%)	41 (67%)	181 (88%)	33 (81%)	21 (10%)	6 (14%)	4 (2%)	2 (5%)
Moderate	69 (23%)	15 (24%)	41 (59%)	8 (54%)	23 (33%)	4 (26%)	5 (8%)	3 (20%)
Bad	22 (9%)	6 (9%)	1 (5%)	1 (17%)	11 (50%)	2 (33%)	10 (45%)	3 (50%)
Total	297	62	223	42	55	12	19	8
Per cent	(100%)	(100%)	(75%)	(68%)	(19%)	(19%)	(6%)	(13%)
ANKLE (All Forms)								
Good	185 (82%)	8 (100%)	156 (84%)	3 (38%)	26 (14%)	5 (62%)	3 (2%)	
Moderate	26 (12%)	...	8 (31%)	...	14 (54%)	...	4 (15%)	
Bad	13 (6%)	...	...	...	1 (8%)	...	12) (92%	
Total	224	8	164	3	41	5	19	
Per cent	(100%)	(100%)	(73%)	(38%)	(18%)	(62%)	(9%)	

TABLE IX.—AVERAGE PERIOD OF DISABILITY

Site of fracture.	All cases.	Average period of disability (months).	Cases terminated under 12 (months).	Average period of disability (months).
Tibia or tibia and fibula, shafts	331	4.7	316	4.2
Ankle	225	4.0	218	3.5
	556*		534	

* 35 extra cases not included in Table VIII are included in Table IX.

per cent in non-operative cases, 75 per cent in operative cases); and conversely, that the worse the anatomical result the worse is function sure to be. Hence it is cause for regret that in the entire series a good anatomical result was secured only in 76

TABLE X.—DOES GOOD ANATOMICAL RESULT LESSEN THE PERIOD OF DISABILITY? FRACTURES OF THE LEG BONES (ALL FORMS)

Anatomical result.	Total.	Under 3 months.	Under 6 months.	Under 9 months.	Under 1 year.	Over 1 year.
Good	335 (79%)	177 (53%)	118 (35%)	30 (9%)	6 (2%)	4 (1%)
Moderate	69 (16%)	15 (22%)	33 (47%)	15 (22%)	2 (3%)	4 (6%)
Bad	20 (5%)	3 (15%)	9 (45%)	3 (15%)	1 (5%)	4 (20%)
	424 (100%)	195 (46%)	160 (38%)	48 (11%)	9 (2%)	12 (3%)

TABLE XI.—GOOD FUNCTIONAL RESULTS FOLLOWING OPERATIVE AND NON-OPERATIVE TREATMENT (SIMPLE FRACTURES)

Site.	American Surgical Association, 1921.		British Medical Association, 1912.	
	Total.	Good function.	Total.	Good function.
Tibia and fibula, shafts	338	239 (70%)	1014	779 (76%)
Ankle fractures	232	188 (81%)	259	127 (49%)
Total leg fractures	570	437 (76%)	1273	906 (71%)

TABLE XII.—GOOD FUNCTIONAL RESULTS FOLLOWING NON-OPERATIVE TREATMENT (SIMPLE FRACTURES)

Site.	American Surgical Association, 1921.		British Medical Association, 1912.	
	Total.	Good function.	Total.	Good function.
Tibia and fibula, shafts	297	206 (70%)	975	748 (76%)
Ankle fractures	224	185 (82%)	255	125 (49%)
Total leg fractures	521	391 (75%)	1230	873 (71%)

TABLE XIII.—GOOD FUNCTIONAL RESULTS FOLLOWING OPERATIVE TREATMENT (SIMPLE FRACTURES)

Site.	American Surgical Association, 1921.		British Medical Association, 1912.	
	Total.	Good function.	Total.	Good function.
Tibia and fibula, shafts	41	33 (82%)	39	31 (79%)
Ankle fractures	8	3 (37%)	4	2 (50%)
Total leg fractures	49	36 (73%)	43	33 (76%)

TABLE XIV.—COMPOUND FRACTURES

	Total.	Non-operative.	Operative.
Fibula, shaft	13	9	4
Tibia, shaft	73	43	30
Tibia and fibula, shafts	206	125	81
Tibia and fibula, ankle	6	5	1
Tibia, ankle	2	1	1
Fibula, ankle	1	1	
	301	184	117

TABLE XV.—COMPOUND FRACTURES

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	167 (58%)	156 (93%)	8 (5%)	3 (2%)
Moderate	83 (29%)	21 (25%)	54 (65%)	8 (10%)
Bad	39 (13%)	3 (8%)	7 (18%)	29 (74%)
	289 (100%)	180 (62%)	69 (24%)	40 (14%)

TABLE XVI.—NON-OPERATIVE CASES (COMPOUND FRACTURES)

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	109 (61%)	102 (93%)	6 (6%)	1 (1%)
Moderate	51 (29%)	15 (29%)	33 (65%)	3 (6%)
Bad	18 (10%)	1 (6%)	3 (17%)	14 (77%)
	178 (100%)	118 (67%)	42 (23%)	18 (10%)

per cent of cases (77 per cent of non-operative and only 69 per cent of operative cases). Should not surgeons be able to secure accurate anatomical reposition of the fragments ("good anatomy") in more than two-thirds of the cases they subject to open reduction?

The influence of age on the functional result is shown in Tables V, VI and VII. These figures emphasize the fact so clearly demonstrated in our earlier tables that in patients under fifteen years of age good function is obtainable with most certainty, and that the older the patient the less certain is good function to be secured. They indicate, moreover, that in patients under fifteen years of age good function is secured in 92 per cent of cases without resort to operation.

In regard to *operative treatment of simple fractures of the leg bones* these tables appear to show that it has proved less easy

TABLE XVII.—OPERATIVE CASES (COMPOUND FRACTURES)

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good . . .	58 (52%)	54 (94%)	2 (3%)	2 (3%)
Moderate . .	32 (29%)	6 (19%)	21 (65%)	5 (16%)
Bad . . .	21 (19%)	2 (10%)	4 (19%)	15 (71%)
	111 (100%)	62 (56%)	27 (24%)	22 (20%)

TABLE XVIII.—INFLUENCE OF AGE ON THE FUNCTIONAL RESULT (COMPOUND FRACTURES)

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Under 15 years:				
Good . .	17 (55%)	15 (88%)	1 (6%)	1 (6%)
Moderate .	9 (29%)	2 (22%)	7 (78%)	
Bad . . .	5 (16%)	1 (20%)	....	4 (80%)
	31 (100%)	18 (58%)	8 (26%)	5 (16%)
Over 15 years:				
Good . .	150 (58%)	141 (94%)	7 (5%)	2 (1%)
Moderate .	74 (29%)	19 (26%)	47 (63%)	8 (11%)
Bad . . .	34 (13%)	2 (6%)	7 (21%)	25 (73%)
	258 (100%)	162 (62%)	61 (24%)	35 (14%)

to secure anatomical reposition of the fragments by operation at any age period (40 to 74 per cent of good anatomical results) than when no operation is done (70 to 90 per cent of good anatomical results); but it must not be overlooked that in the cases treated without operation are included all the subperiosteal and incomplete fractures as well as all those with only trifling displacement in which no reduction of the fragments is required; while in the operative series, almost only, are those fractures with great displacement (overriding, angulation, rotation) in which reduction by non-operative means has usually been tried and has failed before operation was undertaken.

TABLE XIX.—COMPOUND FRACTURES OF THE TIBIA AND THE FIBULA

Anatomical results.	Functional results.							
	Total.		Good.		Moderate.		Bad.	
	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.
SHAFTS (also Shaft of the Tibia alone)								
Good	105 (63%)	58 (53%)	99 (94%)	54 (94%)	6 (6%)	2 (3%)	... ...	2 (3%)
Moderate	46 (27%)	30 (28%)	15 (33%)	6 (20%)	29 (63%)	19 (63%)	2 (4%)	5 (17%)
Bad	17 (10%)	21 (19%)	1 (6%)	2 (10%)	3 (18%)	4 (19%)	13 (76%)	15 (71%)
Total	168	109	115	62	38	25	15	22
Per cent	(100%)	(100%)	(69%)	(57%)	(22%)	(23%)	(9%)	(20%)
ANKLE (All Forms)								
Good	2 (29%)	... ...	1 (50%)	... ...	... ...	... ...	1 (50%)	
Moderate	4 (57%)	2 (100%)	... ...	... ...	3 (75%)	2 (100%)	1 (25%)	
Bad	1 (4%)	... ...	... ...	... ...	... ...	... ...	1 (100%)	
Total	7	2	1	...	3	2	3	
Per cent	(100%)	(100%)	(14%)	...	(43%)	(100%)	(43%)	

One death is recorded from sepsis, five and a half months after open operation and fixation of the tibia by a steel plate.

In Table VIII the anatomical and functional results are analyzed according as the fractures involved the shafts of the leg bones or affected the ankle. Our 1915 statistics clearly showed how much more serious was the prognosis in joint fractures. And in the present table we note that whereas in the case of the shafts of the leg bones bad function follows bad anatomy only in 45 per cent of cases (non-operative), yet in the case of ankle fractures bad anatomical results ensure bad function in 92 per cent of cases. In the shafts of the leg bones a moderate anatomical result secures good function in 58 per cent of cases, whereas in the case of ankle fractures a moderate anatomical result secures good function in less than a third of the cases (31 per cent).

The *average period of disability* in our combined figures (1915 and 1921) shows a slight improvement; in 1915 we found the average period of disability for both shaft and ankle fractures was nearly five months (four and nine-tenths); in the combined statistics, based on 556 cases, this is reduced to four and seven-

TABLE XX.—AVERAGE PERIOD OF DISABILITY (COMPOUND FRACTURES)

Site of fracture.	All cases.	Average period of disability (months).	Cases terminated under 12 (months).	Average period of disability (months).
Tibia or tibia and fibula, shafts	82	7.0	66	5.4
Ankle	8	5.7	8	5.7
	90		74	

TABLE XXI.—DOES GOOD ANATOMICAL RESULT LESSEN THE PERIOD OF DISABILITY? COMPOUND FRACTURES OF THE LEG BONES (ALL FORMS)

Anatomical result.	Total.	Under 3 months.	Under 6 months.	Under 9 months.	Under 1 year.	Over 1 year.
Good . . .	39 (43%)	8 (20%)	21 (54%)	5 (13%)	1 (3%)	4 (10%)
Moderate .	37 (41%)	3 (8%)	15 (39%)	11 (30%)	1 (4%)	7 (19%)
Bad . . .	14 (16%)	....	5 (36%)	2 (14%)	2 (14%)	5 (36%)
	90 (100%)	11 (13%)	41 (45%)	18 (20%)	4 (4%)	16 (18%)

tenths months for fractures of the shafts of the leg bones and to four months only in the case of ankle fractures. Or if only those cases are included in which the period of disability was less than twelve months the figures show an average of four and one-fifth months for the shaft fractures and only three and a half months for the ankle fractures. These later investigations confirm our former conclusions that patients with fractures of the lower extremity whose period of disability exceeds twelve months usually are permanently crippled.

Table X indicates very positively that a good anatomical result lessens the period of disability: 88 per cent of patients with good anatomical results have a period of diability of less than six months; whereas only 69 per cent of those with a moderate anatomical result are able to resume their usual occupations within that space of time.

Comparative Results Shown in American and British Statistics. In Tables XI, XII and XIII are analyzed in parallel columns the results now recorded by the Fracture Committee of the American Surgical Association and those published in 1912 by the Fracture Commission of the British Medical Association. It will be seen that though the latter figures are based on more than twice as many case reports as the American figures, yet that the gross results are sensibly the same, with a small margin to the credit of the American statistics (76 per cent good results compared to 71 per cent good results of the British cases). While the total number of cases of operative treatment is rather too small to make the results very significant (especially in the case of ankle fractures) the great difference in the proportion of good results secured in the non-operative treatment of ankle fractures certainly is worthy of comment. The British statistics show that only 49 per cent of their patients with ankle fractures secured good function, while 82 per cent of American patients recovered without disability; and these figures are the more noteworthy because the number of end-results recorded is very nearly the same in each case (255 British and 224 American). It is possible

that our methods of treatment are more efficient now than those employed by the British a decade ago, and that the interest in fractures aroused in this country by the previous report of this committee is beginning to tell on the end-results; but it seems more likely that in this country more accurate diagnosis has been secured through the more general use of the roentgen ray during the past nine years, and that on this account the American figures include more cases of fracture without displacement than do the British statistics.

Compound Fractures of the Leg Bones. Our tables include 301 cases: 184 non-operative and 117 operative. Good anatomical results were secured only in 58 per cent of cases and good function only in 62 per cent; but of the 167 patients in whom anatomical reposition of the fragments was secured (or in whom no displacement existed) no less than 93 per cent recovered without disability. And, as in the case of simple fractures of the leg bones, it is worthy of comment that even with operative treatment only half the cases (58 out of 111 patients) secured good anatomical results and only a little more than half (56 per cent) secured good function; but that in 54 out of 58 patients, or in 94 per cent of those in whom the surgeon succeeded in obtaining anatomical reposition of the fragments, recovery without any disability is recorded. And the importance of securing accurate anatomical reposition of the fragments is even more apparent in the cases of compound fractures than was the case in simple fractures. In the latter even a moderate anatomical result will allow good function to result in more than half the cases; but in compound fractures only one-quarter of the patients will secure good function unless accurate reduction is secured.

The average period of disability for patients with compound fractures of the shafts of the leg bones is seven months and for those of the ankle five and seven-tenths months. If good anatomical results are obtained, 74 per cent of patients can return to work in less than six months, compared with not more than 47 per cent of patients if only moderate anatomical results are secured.

Conclusions. These investigations emphasize anew the importance of securing anatomical reposition of the fragments not only to secure ultimate good function, but even more to lessen the period of disability. The means of securing reduction naturally vary; in comparatively few cases can gross displacements of the leg bones be corrected by manipulation alone, even with the patient under the influence of a general anesthetic; and such manipulations may do more damage to the soft parts than open operation with direct exposure of the fracture. This objection applies even to reduction by mechanical traction if the latter is employed for rapid reduction. On the other hand *continuous traction* (without the use of an anesthetic except for the primary application of the apparatus) prolonged over a period of several days frequently will secure reduction of overriding and angulation in cases in which attempts at rapid reduction end in failure. Such continuous traction is to be continued after reduction has been secured until the process of union is so far advanced as to prevent recurrence of deformity. This period varies from ten days to three weeks. For such gradual reduction the reporter believes that for fractures of the leg *skeletal traction* by means of the Codivilla or the Steinmann nail is more readily applied and more efficient in the hands of the operating surgeon than is traction secured by means of a Sinclair skate or other indirect apparatus. Skeletal traction is especially indicated in cases of comminuted fractures, simple or compound, in which open fixation of so many fragments would prove difficult or dangerous. In irreducible fractures (whether simple or compound), with two or three fragments only, fixation by a steel plate after open reduction is preferred; but for fractures whose obliquity exceeds twice the diameter of the bone, fixation by encircling bands or wires is more efficient.

Ankle fractures, even more imperatively than fractures of the shafts of the leg bones, require accurate anatomical reduction. The value of the flexed position of the limb in permitting reduction of the fragments (originally advocated by Pott) is not sufficiently appreciated at the present day; nor is the aid to

be derived from tenotomy of the tendon of Achilles. In cases in which one or two well-directed attempts under anesthesia fail to secure reduction of the fracture, open reduction should be done, and in most cases direct fixation of the exposed fragments is requisite.

Finally, the investigations of this committee merely point the way for further improvements in our means of treating fractures of the leg bones. No surgeon having such fractures under his care should rest satisfied until he can publish a series of end-results which in the aggregate will be superior to those recorded in this report, for these are merely the *average*.

FRACTURES OF THE FEMUR

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On account of irregularity in the notes and the absence of much important data a large number of the cases sent in for this report could not be used. Other report sheets were not full; it has required much time and labor to cull from them some of the information the committee set out to ascertain.

There were 584 cases of simple and 70 cases of compound fractures of the femur reported; they were followed up and the final results were ascertained. These cases are used and analyzed and form the basis of the tables. The effort has been made to have the tables full enough and so diversified that the figures themselves will tell the various points one would like to know, especially in regard to the average result of the treatment of fractures of the femur during the last five years.

Unfortunately one of the essential points, namely, method of treatment, has rarely been noted with sufficient accuracy to be made of any great use, except that it is always noted whether a case was treated by the non-operative or closed method or by the operative or open method.

The cases were divided as follows:

Fractures of the neck of the femur	100
Fractures through the trochanters	19
Fractures of the shaft	457
Fractures of the condyles	8
Total	584

There were 448 cases treated by the non-operative method and 136 by the operative method (23.28 per cent of the cases).

Indirect violence in civil life causes 80.6 per cent of all fractures of the femur.

The region involved most frequently in the age period:

One to fifteen years is the middle third.

Fifteen to forty-five years is also the middle third.

Forty-five to sixty years is the neck.

Sixty years and over is also the neck.

Transverse fractures are much more frequent than oblique or other forms of fractures. Spiral and longitudinal fractures of the femur are rare.

The average shortening before reduction was attempted in fractures of the shaft of the femur is 3.5 cm. The average shortening after reduction is 1.5 cm. Shortening after a fracture of the shaft of the femur as much as 2 cm. means the fragments are not apposed end to end; there must be some overlapping. It should be remembered, however, that uninjured femora are frequently of unequal lengths; they not unusually vary as much as 1 cm. Shortening determined by the ordinary methods of measuring extremities is misleading and unreliable. Properly taken skiagrams are much more reliable indices of good anatomical results than shortening. The two methods should both be used to determine the condition. Many cases have demonstrated that as much as 2 cm. shortening by measurements does not prevent good functional results. On the other hand it has been shown that by traction methods the injured extremity may be extended even beyond the measured length of the uninjured member, and yet the fluoroscope will show inaccurate alignment of the fragments. Union in such a position could never rightly be called a good anatomical result. In a series of over 200 cases in which measurements showed no or very little shortening, roentgen-ray examination showed only 2 per cent of accurate anatomical results.

Treatment. It is interesting to note that since the war American surgeons have practically discarded the old Buck's

traction method and are using for the purpose of extension some form of suspension with traction.

A Balkan frame or some modification of this apparatus is the popular one now. Also traction directly on the distal fragment by means of the Steinmann nail, or by some form of tongs, is much more frequently employed than formerly.

The apparatus which proved invaluable in the military service, and which stands out now as the most useful of splints for treating fractures of the femur and humerus, is the Thomas splint. Easily adapted to varying conditions, and efficient in the majority of cases, the Thomas splint is the apparatus which the general practitioner and the surgeon who must treat a fractured shaft of the femur outside a hospital may use with efficiency, and if properly handled, with confident expectation of a good functional result.

Some very late data, obtained from one of the leading hospitals in the United States, are available fortunately for comparison in regard to the improved results of treatment of fractures of the femur. The series of cases is only 57, but is sufficient to bring out some salient points. There were 16 fractures of the neck and 41 fractures of the shaft. The Thomas splint was used in 24 of the cases of fracture of the shaft, sometimes with and sometimes without a Balkan frame. The Balkan frame with suspension-traction was used in 6 cases, and the other 11 cases were variously treated. No Buck extension, however, was used. For comparison the following figures showing the result of the older and newer methods will be interesting and instructive.

In the much larger series of cases treated by the older methods the results were as follows:

NON-OPERATIVE CASES

	Recovered under 6 months.	Recovered over 6 months.
1 to 15 years	83 per cent	17 per cent
15 to 45 "	46 "	54 "
45 to 60 "	53 "	47 "
Over 60 "	14 "	86 "

NEW TREATMENT

	Recovered under 6 months.	Recovered over 6 months.
1 to 15 years	100.0 per cent	
15 to 45 "	28.5 "	71.5 per cent
Over 60 " (no cases).		

It is evident that the result in childhood has been a quicker recovery by the methods of suspension and traction. This may be explained perhaps by the difference in rating "ability to work." In this day of compensation laws the period of disability for adult cases may be reckoned a little longer than formerly. Taken, however, solely as a matter of statistics the newer methods do not produce earlier recovery in the class of men whose usefulness counts most in industrial life. These ratings, be it understood, are for the periods of disability, and in this report period of disability is taken to mean the time from the instant of injury to that of the returning to work.

No surgeon reporting fractures of the femur has noted the use of some efficient form of caliper after union of the fragments when the patient begins to walk about.

Calipers should be used for some weeks in order to prevent bending or other distortion at the seat of fracture as soon as the vertical position and weight-bearing is permitted.

RESULTS OF FRACTURE OF THE FEMUR. These results are rated as anatomical and functional, and good, moderate and bad.

A good anatomical result is taken to mean firm bony union of the well-apposed fragments in the proper axis of the limb.

A good functional result is taken to mean no persistent stiffness of joints or resulting skeletal deformity, and unimpaired ability to do active work or exercise without pain or discomfort.

Table III shows the results of 447 non-operative cases. It will be observed that out of this number there were 180 (42 per cent) good anatomical results, but of these 180 good anatomical results only 157 (87 per cent) proved good functional results: 18 (10 per cent) were moderate and 5 (3 per cent) were positively bad results. Omitting the analysis of the moderate anatomical

results, and going directly to the bad, it will be seen that out of 65 positively bad anatomical results there followed 14 good and 15 moderate functional results, or 44.6 per cent of these bad anatomical results were followed by useful function. The operative cases in Table IV show this point also, but to a less degree.

The results of a fractured femur may be rated in two ways:

1. Scientifically.
2. Industrially.

1. Scientific rating takes in (*a*) the restoration of anatomical integrity and (*b*) restoration of full function of the part.

2. Industrial rating concerns itself solely with restoration of good function.

That these two ratings must necessarily be the same is conclusively disproved by practically all the tables. Reference to Tables III and IV, however, will show the point conclusively.

It is shown by Table III that of 447 cases of fracture of the femur treated by non-operative methods, 180, or 40 per cent, had good anatomical results; 180, or 40 per cent, had moderate anatomical results, and of these 360 cases, 254, or 70.5 per cent, were restored to usefulness, viz.: 157 had good functional results; 58, or 13 per cent, had bad functional results, and 22, or 4.9 per cent, died.

This means that surgeons of the United States and Canada by average non-operative treatment obtain good functional results in 62 per cent of their cases of fracture of the femur in all regions and in all age periods; 23 per cent have moderate functional results—these may be considered as not fully restored but able to do some useful work. The death-rate of this series is a little too high to be representative. This is explained by the fact that these records were culled from over 1000 records altogether and used because the data were full enough to give most of the desired information on the blanks. To check this death-rate another series of over 700 cases was analyzed and the death-rate was found to be 3.69 per cent. This is too low evidently. The true death-rate is probably the mean of these two series

of cases, viz.: about 4 per cent. All these deaths occurred in aged people.

To ascertain the region which when fractured is followed by most deaths an analysis of 131 cases was made as follows:

	Cases.	Deaths.
Fractures of the neck of the femur	31	12
Fractures through the trochanters	7	7
Fractures of the upper one-third of shaft	18	2
Fractures of the middle one-third of shaft	61	2
Fractures of the lower one-third of shaft	14	0
Total	131	23

One must conclude, therefore, that fractures of the upper segments of the femur with the usual concomitant injuries are for aged people very serious injuries indeed.

The causes of death were stated as follows:

	Cases.
Decubitus and sepsis	3
Gangrene and sepsis	1
Shock and exhaustion	16
Pneumonia	2
Pulmonary embolism	1

Operative Treatment. There were 137 operative cases out of a total of 584 cases; 23.28 per cent of the cases of fractures of all regions of the femur were treated by the operative method. Only 15 of the operative cases were fractures involving the extremities of the bone.

There were 7 operations for fractures of the neck.

There were 4 operations for fractures through the trochanters.

There were 4 operations for fractures of the condyles.

There were 121 operations for fractures of the shaft.

Analysis of the cases shows exactly twice as many operations were done for fractures of the middle third of the shaft as for both the upper and lower third together.

Fixation of the bone fragments by some manner of rigid plate was usually the method employed in these cases. Only one death is reported in the series of 136 operative cases; this fatal result was on account of sepsis.

Table IV gives the results in the operative cases. It is shown that though the anatomical results are rated good in 68 per cent of the cases as against 40 per cent in the non-operative method, good functional result is only 77 per cent as compared with 87 per cent of the non-operative cases. Further analysis given in the later tables shows comparatively few cases in the age period one to fifteen years were treated by the operative method; the operative cases were chiefly adult cases. Besides, these cases were operated, in nearly every instance, only because non-operative methods were unsuccessful in obtaining reduction. They were difficult and sometimes complicated cases. It could not reasonably be expected, therefore, that the ultimate result functionally should be as good as the uncomplicated non-operative cases. The interesting point is, however, that of the 94 cases which has good anatomical results, only 73, or 77 per cent, of them obtained good functional results. The moderate functional result ratio is a little higher than the non-operative methods, namely, 23 per cent against 10 per cent. These figures mean that though better anatomical results may be obtained by operative methods, functional results are not as good.

Table XIV shows the average period of disability after operative methods also is a little longer.

Only one death is recorded in this series of 137 operative cases; this occurred from infection. In another series of 146 cases, 2 deaths were reported. Even this is too low as indicative of the results obtained by the average surgeon. The true death-rate following operative treatment of fracture of the femur is about 6 per cent.

One must bear in mind that very few children and no senile cases were operated upon. Individuals in adult life, and presumably in good physical condition, were those treated by the operative method. Good operative recoveries in such cases are to be expected. One marvels, however, that only 1 case of infection was reported in this series of 137 cases.

It is noteworthy that in non-operative cases the functional

results from the moderate anatomical results is much better than in operative cases. In order to obtain good results in operative cases it is very important that good anatomical restitution be obtained. It is shown in Table XV that operative cases have better results when the operation is done soon after the fracture.

The series of 125 cases analyzed seem, however, to show that when the operation is done not later than eight to fourteen days after the injury the results are as good as when the operation is done the first week. *The period of disability is, however, longer after the later operations.*

Period of Disability. The period of disability should be worked out separately for the age period one to fifteen years and for the years of adult life.

In the first place, for purposes of reference and for industrial insurance companies, including compensation boards, the average time of disability for individuals in the working periods of their lives is the one desired to be established. In the second place the time of disability for children cannot be reckoned in the same manner as that for adults. As was stated before, the time of disability in this report is taken to mean the period from the instant of injury to the time the injured person is actually able to return to his former occupation or to some business requiring equal activity and endurance.

Children, as a rule, have no occupation and they are able to go about on their feet and appear restored to full activity at a much earlier period than adults. Therefore, separate classification is made in reckoning periods of disability for the age period one to fifteen years and for all ages beyond fifteen years.

For fractures of the entire femur (including all regions) the time of disability in non-operative cases in the period one to fifteen years is four and one-half months; for all other ages it is eight and one-fifth months. The operative cases show a little longer time of disability, namely, nine months, for adult cases.

For compound fractures of all regions of the femur the average period of disability is eleven months.

TABULATED REPORT ON FRACTURES OF THE FEMUR

TABLE I.—SUMMARY OF ALL CASES

	Total.	Non-operative.	Operative.
Femur, neck	100	93	7
Femur, trochanter	19	15	4
Femur, shaft	457	335	122
Femur, condyles	8	4	4
	584	447	137

TABLE II

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	274 (47%)	228 (83%)	41 (15%)	5 (2%)
Moderate	205 (35%)	101 (49%)	85 (42%)	19 (9%)
Bad	82 (14%)	17 (21%)	22 (26%)	43 (53%)
	561 (96%)	346 (61%)	148 (27%)	67 (12%)
Died	23 (4%)			
	584 (100%)			

TABLE III.—NON-OPERATIVE CASES

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	180 (42%)	157 (87%)	18 (10%)	5 (3%)
Moderate	180 (42%)	97 (54%)	66 (36%)	17 (9%)
Bad	65 (15%)	14 (21%)	15 (23%)	36 (55%)
	425 (99%)	268 (62%)	99 (23%)	58 (13%)
Died	22 (4.9%)			
	447			

TABLE IV.—OPERATIVE CASES

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good	94 (69%)	73 (77%)	21 (23%)	0
Moderate	25 (18%)	8 (32%)	15 (60%)	2 (8%)
Bad	17 (12%)	3 (17%)	4 (24%)	10 (59%)
	136 (99%)	84 (61%)	40 (29%)	12 (10%)
Died	1 (0.6%)			
	137			

TABLE V.—INFLUENCE OF AGE ON THE FUNCTIONAL RESULT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
TOTAL NON-OPERATIVE AND OPERATIVE CASES				
1 to 15 years:				
Good	35 (68%)	32 (91%)	3 (8%)	0
Moderate	14 (27%)	10 (71%)	4 (28%)	0
Bad	2 (3%)	2 (100%)	0	0
	51 (100%)	44 (86%)	7 (13%)	0
Deaths	0			
15 to 45 years:				
Good	15 (50%)	14 (93%)	1 (6%)	0
Moderate	14 (46%)	9 (64%)	4 (28%)	1 (7%)
Bad	1 (3%)	0	0	1 (100%)
	30 (99%)	23 (77%)	5 (16%)	2 (6%)
Deaths	0			
45 to 60 years:				
Good	6 (35%)	6 (100%)	0	0
Moderate	10 (58%)	4 (40%)	6 (60%)	0
Bad	1 (5%	0	0	1 (100%)
	17 (98%)	10 (58%)	6 (36%)	1 (6%
Deaths[1]	5 (22%)			
	22			
Over 60 years:				
Good	2 (20%)	0	1 (50%)	1 (50%)
Moderate	5 (50%)	1 (20%)	4 (80%)	0
Bad	3 (30%)	0	0	3 (100%)
	10 (100%)	1 (10%)	5 (50%)	4 (40%)
Deaths[2]	18 (64%)			
	28			
All over 15 years:				
Good	23 (40%)	20 (87%)	2 (8%)	1 (4%)
Moderate	29 (50%)	14 (48%)	14 (48%)	1 (4%)
Bad	5 (8%)	0	0	5 (100%)
	57 (98%)	34 (59%)	16 (28%)	7 (12%)
Deaths	23 (28%)			
	80			

[1] 3 shock, 1 gangrene, 1 pneumonia. [2] Shock and sepsis.

TABLE VI.—INFLUENCE OF AGE ON NON-OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good . . .	26 (66%)	24 (92%)	1 (3%)	1 (3%)
Moderate .	11 (28%)	9 (81%)	2 (18%)	0
Bad . . .	2 (5%)	2 (100%)	0	0
	39 (99%)	35 (90%)	3 (8%)	1 (2%)
Deaths . .	0			
15 to 45 years:				
Good . . .	11 (46%)	9 (81%)	2 (19%)	0
Moderate .	12 (50%)	11 (91%)	1 (8%)	0
Bad . . .	1 (4%)	0	0	1 (100%)
	24 (100%)	20 (83%)	3 (12%)	1 (4%)
Deaths . .	0			
45 to 60 years:				
Good . .	5 (33%)	5 (100%)	0	0
Moderate .	9 (60%)	5 (55%)	4 (44%)	0
Bad . . .	1 (6%)	0	0	1 (100%)
	15 (99%)	10 (66%)	4 (26%)	1 (6%)
Deaths . .	4 (21%)			
	19			
Over 60 years:				
Good . . .	2 (25%)	0	1 (50%)	1 (50%)
Moderate .	4 (50%)	1 (25%)	3 (75%)	0
Bad . . .	2 (25%)	0	0	2 (100%)
	8 (100%)	1 (12%)	4 (50%)	3 (38%)
Deaths . .	18 (69%)			
	26			
All over 15 years:				
Good . . .	18 (38%)	14 (78%)	3 (16%)	1 (6%)
Moderate .	25 (53%)	17 (68%)	8 (32%)	0
Bad . . .	4 (8%)	0	0	4 (100%)
	47 (99%)	31 (65%)	11 (23%)	5 (10%)
Deaths . .	22 (31%)			
	69			

TABLE VII.—INFLUENCE OF AGE ON OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good	9 (75%)	8 (90%)	1 (10%)	0
Moderate	3 (25%)	1 (33%)	2 (66%)	0
Bad	0	0	0	0
	12 (100%)	9 (75%)	3 (25%)	0
Deaths	0			
15 to 45 years:				
Good	4 (66%)	4 (100%)	0	0
Moderate	2 (33%)	0	2 (100%)	0
Bad	0	0	0	0
	6 (99%)	4 (66%)	2 (33%)	
Deaths	0			
45 to 60 years:				
Good	1 (50%)	1 (100%)	0	0
Moderate	1 (50%)	0	1 (100%)	0
Bad	0	0	0	0
	2 (100%)	1 (50%)	1 (50%)	0
Deaths (sepsis)	1 (33%)			
	3			
Over 60 years:				
Good	0	0	0	0
Moderate	1 (50%)	0	1 (100%)	0
Bad	1 (50%)	0	0	1 (100%)
	2 (100%)	0	1 (50%)	1 (50%)
Deaths	0			
All over 15 years:				
Good	5 (40%)	5 (100%)	0	0
Moderate	4 (50%)	0	4 (100%)	0
Bad	1 (10%)	0	0	1 (100%)
	10 (100%)	5 (50%)	4 (40%)	1 (10%)
Deaths, sepsis	1 (9%)			
	11			

TABLE VIII.—SUMMARY OF FRACTURES OF THE SHAFT OF THE FEMUR, INCLUDING ALL REGIONS

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		NON-OPERATIVE		
Good . . .	152 (46%)	138 (90%)	13 (8%)	1 (0.2%)
Moderate . .	129 (38%)	82 (63%)	42 (33%)	5 (4%)
Bad	51 (16%)	14 (27%)	13 (25%)	24 (48%)
	332 (100%)	234 (70%)	68 (20%)	30 (10%)
		OPERATIVE		
Good . . .	84 (69%)	69 (82%)	15 (18%)	0
Moderate . .	21 (17%)	10 (48%)	9 (43%)	2 (9%)
Bad	16 (14%)	3 (19%)	3 (19%)	10 (62%)
	121 (100%)	82 (67%)	27 (22%)	12 (10%)
		ALL CASES		
Good . . .	236 (52%)	207 (87%)	28 (11%)	1 (0.2%)
Moderate . .	150 (34%)	92 (61%)	51 (34%)	7 (5%)
Bad	67 (14%)	17 (25%)	16 (24%)	34 (50%)
	453 (100%)	316 (69%)	95 (20%)	42 (10%)

Deaths: Non-operative, 3 (1 sepsis, 1 gangrene, 1 decubitus and sepsis). Operative, 1 (sepsis).

TABLE IX.—SUMMARY OF FRACTURES OF THE NECK OF THE FEMUR

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		NON-OPERATIVE		
Good . . .	23 (28%)	14 (60%)	6 (26%)	3 (14%)
Moderate . .	44 (54%)	9 (20%)	25 (57%)	10 (23%)
Bad	14 (18%)	2 (14%)	9 (64%)	3 (22%)
	81 (100%)	25 (30%)	40 (50%)	16 (19%)
		OPERATIVE		
Good . . .	5 (71%)	2 (40%)	3 (60%)	0
Moderate . .	2 (29%)	0	2 (100%)	0
Bad	0	0	0	0
	7 (100%)	2 (29%)	5 (71%)	0
		ALL CASES		
Good . . .	28 (52%)	16 (57%)	9 (33%)	3 (10%)
Moderate . .	46 (32%)	9 (19%)	27 (58%)	10 (23%)
Bad	14 (16%)	2 (14%)	9 (64%)	3 (22%)
	88 (100%)	27 (30%)	45 (51%)	16 (19%)

Deaths: Non-operative, 12 (shock and old age). Deaths: Operative, 0.

TABLE X.—SHOWING COMPARISON BETWEEN NON-OPERATIVE AND OPERATIVE CASES

Anatomical result.		Functional result.							
		Total.		Good.		Moderate.		Bad.	
		Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.	Non-operative.	Operative.
Neck	Good	23	5	14	2	6	3	3	0
	Moderate	44	2	9	0	25	2	10	0
	Bad	14	0	2	0	9	0	3	0
	Died	12							
	Total	93	7	25	2	40	5	16	0
	Per cent.	100	100	30	28	50	71	19	0
Trochanters	Good	4	1	3	1	0	0	1	0
	Moderate	4	2	1	0	2	2	1	0
	Bad	0	1	0	0	0	1	0	0
	Died	7							
	Total	15	4	4	1	2	3	2	0
	Per cent.	100	100	50	25	25	75	25	0
Shaft	Good	152	84	138	69	13	15	1	0
	Moderate	129	21	82	10	42	9	5	2
	Bad	51	16	14	3	13	3	24	10
	Died	3	1						
	Total	335	122	234	82	68	27	30	12
	Per cent.	100	100	70	67	20	22	9	9
Condyles	Good	1	4	1	1	0	3	0	0
	Moderate	3	0	2	0	0	0	1	0
	Bad	0	0	0	0	0	0	0	0
	Total	4	4	3	1	0	3	1	0
	Per cent.	100	100	75	25	0	75	25	0
Entire Femur	Grand total	425	136	266	86	110	38	49	12
	Deaths	22	1						
	Per cent.	100	100	62	63	24	28	11	8

Table XI shows that 37 per cent. of the good anatomical results, 27 per cent. of the moderate anatomical results and 19 per cent. of the bad anatomical results have a disability of less

than six months. It is evident, therefore, that good anatomical results very markedly lessen the period of disability.

TABLE XI.—DOES GOOD ANATOMICAL RESULT LESSEN THE PERIOD OF DISABILITY?

	Anatomical result.	Total.	Period of disability.				
			Under 3 months.	Under 6 months.	Under 9 months.	Under 12 months.	Over 12 months.
Femur (all regions)	Good	178 (50%)	82 (46%)	51 (28%)	9 (6%)	28 (15%)	8 (5%)
	Moderate	122 (34%)	32 (25%)	36 (30%)	17 (14%)	17 (14%)	20 (16%)
	Bad	55 (16%)	9 (16%)	12 (22%)	4 (7%)	3 (6%)	27 (49%)
		355 (100%)	123 (35%)	99 (27%)	30 (8%)	48 (14%)	55 (16%)

Average period of disability for adults, 8.2 months.
Average period of disability for children under fifteen years, 4.5 months.

TABLE XII.—FEMUR, NECK

	Anatomical results.	Total.	Period of disability and functional results.														
			Under 3 months.			Under 6 months.			Under 9 months.			Under 12 months.			Over 12 months.		
			Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.
1 to 15 years	Good	4	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Moderate	2	0	0	1	1	0	0	0	0	0	0	0	0	0	0	1
	Bad	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
15 to 45 years	Good	4	1	0	0	1	0	0	0	0	0	1	1	0	0	0	0
	Good	7	0	0	0	1	0	0	0	0	0	0	0	1	0	0	1
	Bad	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1
45 to 60 years	Good	7	2	0	0	1	0	0	1	0	0	0	2	0	0	1	0
	Moderate	13	3	0	0	0	1	0	0	1	0	1	1	0	0	4	2
	Bad	5	0	0	0	0	0	0	0	0	1	0	0	0	0	0	4
Over 60 years	Good	7	0	1	0	2	1	0	0	0	0	0	0	0	0	1	2
	Moderate	15	0	0	0	0	6	1	0	1	0	0	3	0	0	2	2
	Bad	5	0	0	0	0	1	1	0	0	0	0	0	0	0	0	3
All ages	Good	22	7	1	0	4	1	0	1	0	0	1	3	0	0	2	2
	Moderate	37	3	4	1	2	7	1	0	2	0	1	4	1	0	6	5
	Bad	11	0	0	0	0	1	1	0	0	1	0	0	0	0	0	8

TABLE XIII.—FEMUR, SHAFT

	Anatomical result.	Total.	Period of disability and functional result.														
			Under 3 months.			Under 6 months.			Under 9 months.			Under 12 months.			Over 12 months.		
			Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.
1 to 15 years	Good	89	54	2	0	12	3	0	3	0	0	13	0	0	2	0	0
	Moderate	28	14	4	0	6	0	0	0	0	0	3	0	0	0	1	0
	Bad	10	4	0	4	0	0	1	0	0	0	0	0	0	1	0	0
15 to 45 years	Good	47	9	0	0	19	3	0	3	1	0	10	0	0	1	1	0
	Moderate	37	3	0	0	11	2	0	9	4	1	1	6	0	0	0	0
	Bad	15	0	0	0	5	0	0	3	0	0	0	2	0	1	1	3
45 to 60 years	Good	15	6	0	0	5	0	0	1	0	0	1	1	0	0	1	0
	Moderate	14	0	0	0	4	4	0	0	0	0	2	0	0	0	2	2
	Bad	10	1	0	0	1	2	0	0	0	0	0	0	0	0	0	6
Over 60 years	Good	4	0	0	0	2	0	0	0	0	0	1	0	0	0	1	0
	Moderate	7	1	0	0	1	0	0	0	0	0	1	1	0	0	2	1
	Bad	9	0	0	0	0	0	1	0	0	0	0	1	0	0	1	6
All ages	Good	155	69	2	0	38	6	0	7	1	0	25	1	0	3	3	0
	Moderate	86	18	4	0	22	6	0	9	4	1	7	7	0	0	5	3
	Bad	44	5	0	4	6	2	2	3	0	0	0	3	0	2	2	15

Average period of disability for adult cases, 8.2 months.
Average period of disability for children one to fifteen years, 4.5 months.

TABLE XIV.—FEMUR, SHAFT. OPERATIVE COMPARED WITH NON-OPERATIVE TREATMENT

Age.	Number of cases.		Period of disability.				
			Under 3 mos.	Under 6 mos.	Under 9 mos.	Under 12 mos.	Over 12 mos.
1 to 15 years	127	Non-operative . . 99 (78%)	66 (67%)	16 (16%)	0	16 (16%)	1 (1%)
		Operative 28 (22%)	17 (62%)	4 (14%)	3 (10%)	1 (4%)	3 (10%)
15 to 45 years	99	Non-operative . . 66 (67%)	6 (9%)	24 (37%)	16 (24%)	16 (24%)	4 (6%)
		Operative 33 (33%)	6 (18%)	16 (48%)	5 (15%)	4 (13%)	2 (6%)
45 to 60 years	39	Non-operative . . 31 (79%)	7 (23%)	10 (30%)	4 (13%)	3 (11%)	7 (23%)
		Operative 8 (21%)	2 (25%)	1 (12%)	0	1 (12%)	4 (50%)
Over 60 years	20	Non-operative . . 16 (80%)	1 (7%)	1 (7%)	0	3 (18%)	11 (68%)
		Operative 4 (20%)	0	3 (75%)	0	1 (25%)	0
All ages	285	Non-operative . . 212 (75%)	80 (38%)	51 (24%)	20 (9%)	38 (18%)	23 (10%)
		Operative 73 (25%)	25 (35%)	24 (33%)	8 (11%)	7 (9%)	9 (12%)

Deaths: Non-operative, 4; operative, 0.
Average period of disability for non-operative adult cases, 8.2 months.
Average period of disability for operative adult cases, 9.0 months.

TABLE XV.—INFLUENCE ON RESULT OF TIME ELAPSING BETWEEN INJURY AND OPERATION (FEMUR)

Ages, years.			Date of operation, days.				Anatomical position.	Functional result.		
1 to 15	15 to 45	45 and over.	0 to 7	8 to 14	15 to 21	22 to 28		Good.	Mod.	Bad.
48	..	..	17	..	..	..	Good . . . 16	16		
							Moderate . 1	..	1	
							Bad . . . 0	..	..	0
				16	..	..	Good . . . 15	15		
							Moderate . 1	..	1	
							Bad . . . 0	..	..	0
					7	..	Good . . . 7	7		
							Moderate . 0	..	0	
							Bad . . . 0	..	..	0
						8	Good . . . 0	0		
							Moderate . 8	..	8	
							Bad . . . 0	..	..	0
	50	..	..	18	..	..	Good . . . 17	17		
							Moderate . 1	..	1	
							Bad . . . 0	..	..	0
					3	..	Good . . . 2	2		
							Moderate . 1	..	1	
							Bad . . . 0	..	..	0
						29	Good . . . 1	1		
							Moderate . 26	..	26	
							Bad . . . 2	..	..	2
		27	..	11	..	..	Good . . . 5	5		
							Moderate . 16	..	6	
							Bad . . . 0	..	..	0
						16	Good . . . 0	0		
							Moderate . 12	..	12	
							Bad . . . 4	..	..	4
125 cases		..	17	45	10	53	Good . . . 63	63		
							Moderate . 56	..	56	
							Bad . . . 6	..	..	6

TABLE XVI.—SUMMARY OF SIMPLE FRACTURES OF THE FEMUR IN THE SEVERAL REGIONS

Femur.	Number of cases.	Non-operative.	Operative.
Neck	31	30	1
Upper third	18	13	5
Middle third	61	47	14
Lower third	14	11	3
Through trochanter	7	7	0
	131	108	23
Deaths—Neck	12	12	0
Upper third	2	1	1
Middle third	2	2	0
Lower third	0	0	0
Through trochanter	7	7	0
	23	22	1

Tables XVI to XXIV are the result of a study of 131 fractures of the femur to show the number of cases and comparative results of non-operative and operative treatment of fractures in the several regions of the femur. Non-operative cases, 108; operative cases, 23.

The middle third shows by far the largest number of cases. That the neck is fractured very rarely except in senile individuals the tables show conclusively.

Fractures of the femur are more frequent in the age period 1 to 15 years in all regions except the neck and trochanter.

In this series of cases, except in very old individuals, good anatomical results in cases of fracture, in all regions, were followed by 100 per cent. good functional results in non-operative cases except in the lower third.

The period of disability is a little less in non-operative cases. One must remember, however, that the operative method is used when the non-operative is not practicable or does not suffice to reduce or retain the fracture. Operative cases are therefore essentially more difficult cases and the condition for rapid union are not as good; however, the operation makes a

simple fracture a compound one. The trauma of the soft tissues, and especially the forcible manipulation of the fragments, undoubtedly retards bony union.

Except two the deaths all occurred in senile cases and from fractures of the upper segments of the bone. This emphasizes the fact that fractures of the femur anywhere above the middle third are very serious injuries for aged patients.

TABLE XVII.—COMPARATIVE RESULTS OF OPERATIVE AND NON-OPERATIVE TREATMENT OF SIMPLE FRACTURES OF THE FEMUR IN THE SEVERAL REGIONS

Anatomical result.	Total.	Good.	Functional result. Moderate.	Bad.
NECK:		NON-OPERATIVE		
Good	7 (23%)	4 (57%)	2 (28%)	1 (14%)
Moderate	8 (26%)	4 (50%)	4 (50%)	0
Bad	3 (10%)	0	0	3 (100%)
Deaths	12 (40%)			
	30 (99%)	8 (50%)	6 (25%)	4 (23%)
NECK:		OPERATIVE		
Good	0	0	1 (100%)	0
Moderate	1 (100%)	0	0	0
Bad	0	0	0	0
Deaths	0			
	1 (100%)	0	1 (100%)	0

TABLE XVIII

Anatomical result.	Total.	Good.	Functional result. Moderate.	Bad.
UPPER THIRD:		NON-OPERATIVE		
Good	6 (46%)	5 (82%)	1 (17%)	0
Moderate	6 (46%)	5 (82%)	1 (17%)	0
Bad	0	0	0	0
Deaths	1 (8%)			
	13 (100%)	10 (83%)	2 (16%)	0
UPPER THIRD:		OPERATIVE		
Good	3 (60%)	2 (66%)	1 (33%)	0
Moderate	1 (20%)	1 (100%)	0	0
Bad	0	0	0	0
Deaths	1 (20%)			
	5 (100%)	3 (75%)	1 (25%)	0

TABLE XIX

Anatomical result.	Total.	Good.	Functional result. Moderate.	Bad.
MIDDLE THIRD:		NON-OPERATIVE		
Good	25 (53%)	25 (100%)	0	0
Moderate	18 (38%)	12 (66%)	5 (27%)	1 (6%)
Bad	2 (4%)	2 (100%)	0	0
Deaths	2 (4%)			
	47 (99%)	39 (86%)	5 (12%)	1 (25%)
MIDDLE THIRD:		OPERATIVE		
Good	9 (64%)	9 (100%)	0	0
Moderate	5 (35%)	2 (40%)	3 (60%)	0
Bad	0	0	0	0
Deaths	0			
	14 (99%)	11 (78%)	3 (22%)	0

TABLE XX

Anatomical result.	Total.	Good.	Functional result. Moderate.	Bad.
LOWER THIRD:		NON-OPERATIVE		
Good	6 (54%)	5 (82%)	1 (17%)	0
Moderate	4 (36%)	3 (75%)	1 (25%)	0
Bad	1 (10%)	0	0	1 (100%)
Deaths	0			
	11 (100%)	8 (72%)	2 (18%)	1 (9%)
LOWER THIRD:		OPERATIVE		
Good	2 (66%)	2 (100%)	0	0
Moderate	0	0	0	0
Bad	1 (33%)	0	0	1 (100%)
Deaths	0			
	3 (99%)	2 (66%)	0	1 (33%)

TABLE XXI.—INFLUENCE OF AGE PERIOD IN THE DIFFERENT REGIONS

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
NECK:		NON-OPERATIVE		
1 to 15 years:				
Good . .	0	0	0	0
Moderate .	1 (100%)	1 (100%)	0	0
Deaths .	0	0	0	0
	1 (100%)	1 (100%)	0	0
NECK:		NON-OPERATIVE		
15 to 45 years:				
Good . .	2 (100%)	2 (100%)	0	0
Moderate .	0	0	0	0
Bad . .	0	0	0	0
Deaths .	0			
	2 (100%)	2 (100%)	0	0
NECK:		OPERATIVE		
15 to 45 years: No cases reported.				
NECK:		NON-OPERATIVE		
45 to 60 years:				
Good . .	3 (30%)	3 (100%)	0	0
Moderate .	4 (40%)	2 (80%)	2 (50%)	0
Bad . .	1 (10%)	0	0	1 (100%)
Deaths .	2 (20%)	0	0	0
	10 (100%)	5 (63%)	2 (25%)	1 (12%)
Deaths: 2 pneumonia.				
NECK:		OPERATIVE		
45 to 60 years:				
Good . .	0	0	0	0
Moderate .	1 (100%)	0	1 (100%)	0
Bad . .	0	0	0	0
Deaths .	0			
	1 (100%)	0	1 (100%)	0
NECK:		NON-OPERATIVE		
Over 60 years:				
Good . .	2 (12%)	0	1 (50%)	1 (50%)
Moderate .	3 (17%)	0	3 (100%)	0
Bad . .	2 (12%)	0	0	2 (100%)
Deaths[1] .	10 (58%)			
	17 (99%)	0	4 (56%)	3 (43%)
NECK:		OPERATIVE		
Over 60 years: No cases reported.				

[1] Deaths from old age and shock.

TABLE XXII.

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		NON-OPERATIVE		
UPPER THIRD:				
1 to 15 years:				
Good	5 (71%)	5 (100%)	0	0
Moderate	2 (28%)	1 (50%)	1 (50%)	0
Bad	0	0	0	0
Deaths	0			
	7 (99%)	6 (85%)	1 (14%)	0

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		OPERATIVE		
UPPER THIRD:				
1 to 15 years:				
Good	0	0	0	0
Moderate	1 (100%)	0	1 (100%)	0
Bad	0	0	0	0
Deaths	0			
	1 (100%)	0	1 (100%)	0
		NON-OPERATIVE		
UPPER THIRD:				
15 to 45 years:				
Good	1 (33%)	1 (100%)	0	0
Moderate	2 (66%)	2 (100%)	0	0
Bad	0	0	0	0
Deaths	0			
	3 (99%)	3 (100%)	0	0
		OPERATIVE		
UPPER THIRD:				
15 to 45 years:				
Good	2 (100%)	2 (100%)	0	0
Moderate	0	0	0	0
Bad	0	0	0	0
Deaths	0			
	2 (100%)	2 (100%)	0	0

NON-OPERATIVE

UPPER THIRD:				
45 to 60 years:				
Good . .	0	0	0	0
Moderate .	1 (50%)	1 (100%)	0	0
Bad . .	0	0	0	0
Deaths[1] .	1 (50%)			
	2 (100%)	1 (100%)	0	0

OPERATIVE

UPPER THIRD:				
45 to 60 years:				
Good . .	1 (100%)	1 (100%)	0	0
Moderate .	0	0	0	0
Bad . .	0	0	0	0
Deaths .	0			
	1 (100%)	1 (100%)	0	0

NON-OPERATIVE

UPPER THIRD:				
Over 60 years:				
Good . .	0	0	0	0
Moderate .	1 (50%)	1 (100%)	0	0
Bad . .	0	0	0	0
Deaths[2] .	1			
	1 (50%)	1 (100%)	0	0

OPERATIVE

UPPER THIRD:
Over 60 years: No cases reported.

[1] Sepsis, 1. [2] Diabetes.

TABLE XXIII

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		NON-OPERATIVE		
MIDDLE THIRD:				
1 to 15 years:				
Good . .	19 (61%)	19 (100%)	0	0
Moderate .	8 (26%)	5 (51%)	3 (38%)	0
Bad . .	2 (6%)	2 (100%)	0	0
Deaths[1] .	2 (6%)			
	31 (99%)	26 (89%)	3 (11%)	0
		OPERATIVE		
MIDDLE THIRD:				
1 to 15 years:				
Good . .	8 (60%)	8 (100%)	0	0
Moderate .	2 (40%)	1 (50%)	1 (50%)	0
Bad . .	0	0	0	0
Deaths .	0			
	10 (100%)	9 (90%)	1 (10%)	0
		NON-OPERATIVE		
MIDDLE THIRD:				
15 to 45 years:				
Good . .	5 (38%)	5 (100%)	0	0
Moderate .	8 (61%)	5 (62%)	2 (25%)	1 (13%)
Bad . .	0	0	0	0
Deaths .	0			
	13 (99%)	10 (76%)	2 (15%)	1 (8%)
		OPERATIVE		
MIDDLE THIRD:				
15 to 45 years:				
Good . .	1 (20%)	1 (100%)	0	0
Moderate .	2 (40%)	0	2 (100%)	0
Bad . .	0	0	0	0
Deaths[1] .	2 (40%)			
	5 (100%)	1 (33%)	2 (66%)	0

[1] 1 gangrene, 1 sepsis.

[1] 1 gangrene, 1 shock.

NON-OPERATIVE

MIDDLE THIRD:				
45 to 60 years:				
Good . .	1 (20%)	1 (100%)	0	0
Moderate .	2 (40%)	0	2 (100%)	0
Bad . .	0	0	0	0
Deaths[1] .	2 (40%)			
	5 (100%)	1 (33%)	2 (66%)	0

OPERATIVE

MIDDLE THIRD:				
45 to 60 years:				
Good . .	0	0	0	0
Moderate .	0	0	0	0
Bad . .	0	0	0	0
Deaths .	0	0	0	0
	0	0	0	0

NON-OPERATIVE

MIDDLE THIRD:
Over 60 years: No cases reported.

OPERATIVE

MIDDLE THIRD:				
Over 60 years:				
Good . .	0	0	0	0
Moderate .	1 (100%)	0	1 (100%)	0
Bad . .	0	0	0	0
	1 (100%)	0	1 (100%)	0

TABLE XXIV

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		NON-OPERATIVE		
LOWER THIRD:				
1 to 15 years:				
Good . .	2 (100%)	1 (50%)	1 (50%)	0
Moderate .	0	0	0	0
Bad . .	0	0	0	0
Deaths .	0			
	2 (100%)	1 (50%)	1 (50%)	0
		OPERATIVE		
LOWER THIRD:				
1 to 15 years:				
Good . .	1 (100%)	1 (100%)	0	0
Moderate .	0	0	0	0
Bad . .	0	0	0	0
Deaths .	0			
	1 (100%)	1 (100%)	0	0
		NON-OPERATIVE		
LOWER THIRD:				
15 to 45 years:				
Good . .	3 (50%)	3 (100%)	0	0
Moderate .	2 (33%)	1 (50%)	1 (50%)	0
Bad . .	1 (17%)	0	0	1 (100%)
Deaths .	0	0	0	0
	6 (100%)	4 (66%)	1 (16%)	1 (16%)
		OPERATIVE		
LOWER THIRD:				
15 to 45 years:				
Good . .	1 (100%)	1 (100%)	0	0
Moderate .	0	0	0	0
Bad . .	0	0	0	0
Deaths .	0			
	1 (100%)	1 (100%)	0	0

NON-OPERATIVE

LOWER THIRD:				
45 to 60 years:				
Good . .	1 (33%)	1 (100%)	0	0
Moderate .	2 (66%)	1 (50%)	1 (50%)	0
Bad . .	0	0	0	0
Deaths .	0	0	0	0
	3 (99%)	2 (66%)	1 (33%)	0

OPERATIVE

LOWER THIRD:
45 to 60 years: No cases reported.

TABLE XXV

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
NON-OPERATIVE				
LOWER THIRD:				
Over 60 years: No cases reported.				
OPERATIVE				
LOWER THIRD:				
Over 60 years:				
Good . .	0	0	0	0
Moderate .	0	0	0	0
Bad . .	1 (100%)	1 (100%)	0	0
Deaths .	0	0	0	0
	1 (100%)	1 (100%)	0	0

TIME ELAPSING BETWEEN INJURY AND EFFICIENT TREATMENT. In a series of 248 cases of fracture of the femur, 107 were treated within the first twelve hours; 31 between thirteen and twenty-four hours; 20 between twenty-five and forty-eight hours; 14 between forty-nine and seventy-two hours; 13 after seventy-three hours. In 63 cases the time was not stated.

From a collection of 165 cases of fracture of the femur which were operated on it was possible to analyze more or less accurately a series of 125 cases and thus learn whether or not prompt attention markedly affected their results: 48 were children up to fifteen years; 17 were operated on within seven days, 16

secured normal function and 1 moderate function; 16 were operated on within fourteen days, 15 secured normal function and 1 moderate function; 7 were operated on within twenty-one days and secured normal function; 8 were operated on after twenty-eight days and secured moderate function. Of the whole 48 patients the results were 80 per cent. good function and 20 per cent. moderate function.

There were 50 cases between fifteen and forty-five years: 18 were operated on within fourteen days, 17 secured normal function and 1 moderate function; 3 were operated on within twenty-one days, 2 secured normal function and 1 moderate function; whereas 29 were operated on later and secured only normal function in 1, 26 moderate, and 2 bad function. Of the whole number, 50 patients, 40 per cent. secured normal function, 56 per cent. secured moderate function, and 4 per cent. secured bad function.

There were 27 cases over forty-five years of age: 5 only secured normal function and all were operated on within fourteen days; 6 additional cases operated on within fourteen days secured only moderate function; whereas 16 cases were operated on later than fourteen days and secured only moderate function in 12 cases and bad function in 4 cases. Of the whole number, 27 patients, 18 per cent. secured normal function, 67 per cent. moderate function, and 15 per cent. bad function.

TABLE XXVI.—SUMMARY OF ALL CASES OF COMPOUND FRACTURES OF THE FEMUR

Anatomical result.	Total.	Non-operative.	Operative.
Neck	1	1	0
Upper third	10	6	4
Middle third	20	7	13
Lower third	39	10	29
	70	24	46

TABLE XXVII

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
Good . . .	42 (66%)	38 (90%)	4 (10%)	0
Moderate . .	10 (16%)	7 (70%)	3 (30%)	0
Bad	11 (18%)	0	9 (82%)	2 (18%)
	63 (100%)	45 (71%)	16 (25%)	2 (4%)
Deaths . . .	7			
	70			

TABLE XXVIII

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
		NON-OPERATIVE		
Good . . .	16 (76%)	15 (93%)	1 (7%)	0
Moderate . .	4 (20%)	2 (50%)	2 (50%)	0
Bad	1 (4%)	0	0	1 (100%)
	21	17 (81%)	3 (14%)	1 (5%)
Deaths . . .	3			
	24			

TABLE XXIX

Anatomical result	Total..	Functional result. Good.	Moderate.	Bad.
		OPERATIVE		
Good . . .	26 (62%)	25 (97%)	1 (3%)	0
Moderate . .	6 (14%)	5 (83%)	1 (17%)	0
Bad	10 (24%)	0	2 (20%)	8 (80%)
	42 (100%)	30 (73%)	4 (9%)	8 (18%)
Deaths . . .	4			
	46			

TABLE XXX.—INFLUENCE OF AGE ON THE FUNCTIONAL RESULT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good . .	8 (66%)	8 (100%)	0	0
Moderate .	1 (8%)	1 (100%)	0	0
Bad . . .	3 (25%)	0	3 (100%)	0
	12 (99%)	9 (75%)	3 (25%)	0
Deaths, 3 (shock).				
15 to 45 years:				
Good . .	32 (73%)	31 (97%)	1 (3%)	0
Moderate .	8 (18%)	5 (62%)	2 (25%)	1 (13%)
Bad . . .	4 (9%)	0	0	4 (100%)
	44 (100%)	36 (82%)	3 (7%)	5 (11%)
Deaths, 1 (loss of blood).				
45 to 60 years:				
Good . .	2 (33%)	2 (100%)	0	0
Moderate .	1 (16%)	0	1 (100%)	0
Bad . . .	3 (50%)	0	1 (33%)	2 (66%)
	6 (99%)	2 (33%)	2 (33%)	2 (66%)
Deaths, 2 (shock).				
Over 60 years:				
Good . .	1 (100%)	1 (100%)	0	0
Moderatef .	0	0	0	0
Bad . . .	0	0	0	0
	1 (100%)	1 (100%)	0	0
Deaths, 1 (shock).				

Total deaths, 7 (6 shock, 1 loss of blood).

TABLE XXXI.—INFLUENCE OF AGE ON NON-OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good . .	2 (66%)	2 (100%)	0	0
Moderate .	1 (33%)	1 (100%)	0	0
Bad . . .	0	0	0	0
	3 (90%)	3 (100%)	0	0
Deaths, 2 (shock).				

15 to 45 years:				
Good . .	11 (78%)	11 (100%)	0	0
Moderate .	3 (22%)	0	2 (66%)	1 (33%)
Bad . . .	0	0	0	0
	14 (100%)	11 (78%)	2 (14%)	1 (8%)
45 to 60 years:				
Good . .	2 (66%)	2 (100%)	0	0
Moderate .	0	0	0	0
Bad . . .	1 (33%)	0	0	1 (100%)
	3 (100%)	2 (66%)	0	1 (33%)
Over 60 years:				
Good . .	1 (100%)	1 (100%)	0	0
Moderate .	0	0	0	0
Bad . . .	0	0	0	0
	1 (100%)	1 (100%)	0	0

Deaths, 1 (shock). Total deaths, 3 (shock).

TABLE XXXII.—INFLUENCE OF AGE ON OPERATIVE TREATMENT

Anatomical result.	Total.	Functional result. Good.	Moderate.	Bad.
1 to 15 years:				
Good . .	6 (66%)	6 (100%)	0	0
Moderate .	0	0	0	0
Bad . . .	3 (33%)	0	3 (100%)	0
	9 (99%)	6 (66%)	3 (33%)	0

Deaths, 1 (shock).

15 to 45 years:				
Good . .	21 (70%)	20 (95%)	1 (5%)	0
Moderate .	5 (16%)	3 (60%)	2 (40%)	0
Bad . . .	4 (14%)	0	0	4 (100%)
	30 (100%)	23 (76%)	3 (10%)	4 (14%)

Deaths, 1 (loss of blood).

45 to 60 years:				
Good . .	0	0	0	0
Moderate .	1 (33%)	0	1 (100%)	0
Bad . . .	2 (66%)	0	1 (50%)	1 (50%)
	3 (99%)	0	2 (66%)	1 (33%)

Deaths, 2 (shock).

Over 60 years: No cases reported. Total deaths, 4 (3 shock, 1 loss of blood.

TABLE XXXIII.—COMPOUND FRACTURES OF THE FEMUR (SHAFT)

Anatomical result.	Total.	Good.	Functional result. Moderate.	Bad.
		NON-OPERATIVE		
Good . . .	15 (75%)	15 (100%)	0	0
Moderate . .	4 (20%)	2 (50%)	2 (50%)	0
Bad	1 (5%)	0	0	1 (10%)
	20 (100%)	17 (85%)	2 (10%)	1 (5%)
Deaths, 3				
		OPERATIVE		
Good . . .	26 (62%)	25 (97%)	1 (3%)	0
Moderate . .	6 (14%)	5 (83%)	1 (17%)	0
Bad	10 (24%)	0	2 (20%)	8 (80%)
	42 (100%)	30 (73%)	4 (9%)	8 (18%)
Deaths, 4.				

TABLE XXXIV.—COMPOUND FRACTURES OF THE FEMUR (NECK)

Anatomical result.	Total.	Good.	Functional result. Moderate.	Bad.
		NON-OPERATIVE		
Good . . .	1 (100%)	1 (100%)	0	0
Moderate . .	0	0	0	0
Bad	0	0	0	0
	1 (100%)	1 (100%)	0	0
		OPERATIVE		

No cases reported.

TABLE XXXV.—DOES GOOD ANATOMICAL RESULT LESSEN THE PERIOD OF DISABILITY

COMPOUND FRACTURES OF THE FEMUR

Anatomical result.	Total.	Period of disability.				
		Under 3 mos.	Under 6 mos.	Under 9 mos.	Under 12 mos.	Over 12 mos.
FEMUR (all forms):						
Good . . .	3 (25%)	1 (33%)	1 (33%)	1 (33%)	0	0
Moderate . .	7 (58%)	0	2 (28%)	4 (57%)	0	1 (15%)
Bad	2 (17%)	0	0	1 (50%)	0	1 (50%)
	12 (100%)	1 (8%)	3 (25%)	6 (50%)	0	2 (16%)

NOTE.—Table XXXV represents the only cases reported the last year with periods of disability noted. These combined with those of the 1915 report, namely, 51 cases, would give 63 cases, an average disability of 11.5 months. The one case with a noted disability under three months was a child eight years of age. For adult cases, therefore, the average disability proves to be 11.8 months.

TABLE XXXVI—COMPOUND FRACTURES OF THE FEMUR, ALL FORMS

	Anatomical result.	Total.	Period of disability and functional result.														
			Under 3 months.			Under 6 months.			Under 9 months.			Under 12 months.			Over 12 months.		
			Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.	Good.	Mod.	Bad.
1 to 15 years	Good	1	1	0	0	0	0	0	0	0	0						
	Moderate	1	0	0	0	1	0	0	0	0	0						
	Bad	0	0	0	0	0	0	0	0	0	0						
15 to 45 years	Good	2	0	0	0	1	0	0	0	1	0						
	Moderate	5	0	0	0	1	0	0	3	1	0						
	Bad	1	0	0	0	0	0	0	0	0	1						
45 to 60 years	Good	0	0	0	0	0	0	0	0	0	0						
	Moderate	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
	Bad	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
Over 60 years	No cases.																

TABLE XXXVII.—OPERATIVE COMPARED WITH NON-OPERATIVE TREATMENT

COMPOUND FRACTURES OF THE FEMUR

Age.	Number of cases.	Period of disability.				
		Under 3 mos.	Under 6 mos.	Under 9 mos.	Under 12 mos.	Over 12 mos.
1 to 15 years	2 { Non-operative . . 1 (50%)	0	1 (100%)	0	0	0
	{ Operative 1 (50%)	1 (100%)	0	0	0	0
15 to 45 years	8 { Non-operative . . 3 (37%)	0	2 (66%)	1 (33%)	0	0
	{ Operative 5 (61%)	0	0	5 (100%)	0	
45 to 60 years	2 { Non-operative . . 0	0	0	0	0	0
	{ Operative 2 (100%)	0	0	0	0	2 (100%)
Over 60 years	No cases.					
All ages	12 { Non-operative . . 4 (33%)	0	3 (75%)	1 (25%)	0	0
	{ Operative 8 (66%)	1 (12%)	0	5 (61%)	0	2 (25%)

NOTE.—Table XXXVIII records only cases reported the last year and represents the latest after-the-war treatment. It is too small a number to have conclusive value, but is suggestive of what may be expected in regard to periods of disability. The two cases which went over twelve months had each of them twenty months' disability.

TABLE XXXVIII.—COMPARATIVE RESULTS OF OPERATIVE AND NON-OPERATIVE CASES

Compound Fractures of the Femur

Fracture.	Operative cases.			Non-operative cases.		
	Cases.	Good anatomical result.	Good functional result.	Cases.	Good. anatomical result.	Good functional result.
Femur Shaft	46	57%	65%	23	66%	69%

CERTAIN PROBLEMS CONCERNING FRACTURES OF BONE

By CHARLES L. SCUDDER, M.D.
BOSTON, MASS.

Our problems today have been the problems of the ages. Listen to what the wise Greek surgeon, Hippocrates, said in 400 B.C. in writing of fractures:

"I know physicians who have the reputation of being skilled in giving the proper positions to the arm in binding it up after fracture, while in reality they are only showing their ignorance. But many other things in our art are judged of in this manner, for people rather admire what is new, although they do not know whether it be proper or not, than what they are accustomed to and know already to be proper; and what is strange, they prefer to what is obvious." "And," Hippocrates continues, "I must now state what the mistakes of medical men are, which I wish to unteach," etc., etc.

Under the term "Fractures of Bone" I would eventually, when ideal conditions obtain, include the following:

Fracture of the skull, protector of the brain.

Fracture of the spine, so adequately shielding the cord from injury.

Fracture of the thorax, with possible damage to the contained pleura, lung and heart.

Fracture of the pelvis, containing abdominal organs sometimes seriously damaged.

Fracture of the long and short bones of the upper and lower extremities.

Fracture of the articular surfaces of all joints.

Gunshot fractures of the skeleton.

Open or compound fractures, potentially infected wounds.

All dislocations.

It will not be forgotten that associated with these fractures there may be contused and lacerated wounds, and there may be sprains of joints distant from the apparent injury. Shock may be present, slight or serious. In addition, damage to muscles, to single nerves or nerve plexuses, to tendons, and to important bloodvessels may complicate the situation.

There are certain problems necessarily included in this group of injuries which are not altogether settled, viz.:

(*a*) The process of repair of fractures.

(*b*) The causes of ununited fractures.

(*c*) The treatment of ununited fractures.

(*d*) The repair of pathological fractures.

(*e*) The proper handling of crushed fractures.

(*f*) The treatment of malunited fractures.

This conception of the surgery of fractures, therefore, is seen to cover pretty completely traumatic surgery. A complete general surgical training and the ability to exercise that sound judgment which comes with experience are obviously necessary for the man who is to handle well the many difficult situations which may arise in any individual case. The task is a large one.

FRACTURES OF THE LONG BONES

The older methods (rigid-bone methods) of treating fractures are: By "setting" the fractured bone; by the use of splints and plaster-of-Paris for fixing the "set bones;" by holding the joints immobilized above and below the fracture; by keeping tightly constricting and compressing splints and bandages on the fractured limb for weeks, the soft parts, the skin, nerves, vessels and muscles all being left without care. These methods are abominable and should not be tolerated today. Good results under this ancient regimen did occur—often obtained in spite of treatment not because of it.

The principles underlying present methods of handling fractures of the long bones:

The French massage treatment advocated, practised and taught in cases of fracture by Lucas Championiére and his pupils, notably Mennell in London;

The extension and counterextension method advocated by Bardenheuer, of Cologne, and consistently followed by several English surgeons (notably Sir Robert Jones) and by American surgeons. Extension and counterextension, thus used, requires careful supervision, a nice discrimination in its application, and permits supported active movements of the joints adjacent to the fractured bone at an early date and continuously throughout treatment;

The direct bony traction of Codivilla in Italy, of Steinman in Switzerland, of Ransohoff in America, bringing most effectively and accurately the forces of traction and countertraction into action;

The direct grasp of a bone without penetration for the application of traction advocated by Hey-Groves and others;

The direct traction methods of Parkhill and Freeman in America;

The direct operative treatment advocated and popularized by Sir Arbuthnot Lane and others here and abroad;

The suspension of fractures—helping to eliminate muscular contraction and rendering more effective traction forces;

The use of the roentgen ray.

All these principles and methods underlying fracture treatment have been recognized for years. Recently they have had renewed attention focussed upon them by many surgeons because of the dire necessities of war. The treatment of war injuries tends to simplicity. Consequently many irrelevant forms of apparatus have been eliminated.

The war has further influenced the treatment of fractures by simplifying the emergency treatment; by an effective treatment of compound fractures (through the Carrel-Dakin principles); by eliminating certain inefficient traditional methods of

treatment and by emphasizing the possibilities of suspension and direct traction scarcely dreamed of a few years ago.

Experience, too, has gradually decreased the instances in which foreign unabsorbable material should be employed. The operative treatment is better defined in the selection of suitable cases. The selection of the appropriate non-operative treatment in individual cases can be more accurately made. The old principles of prolonged fixation of joints adjacent to fractures has passed. The early active motion of joints under protection is employed and is furnishing remarkable results.

The results of the treatment of fractures of the long bones today are unusual compared with the results under the older methods.

General surgeons are, as a group, uninterested in treating fractures. There are, of course, exceptions in every community. The attention of the general surgeon has been directed from fractures to the more dramatic fields of abdominal surgery.

In general, the collective results of fracture treatment are even now throughout the country deplorably poor. The community, every fracture patient, the workingman in particular, are all asking for better results. The employer of labor is demanding that injured men be gotten back to work more quickly, that fewer hours be lost. Great pressure is being brought to bear on the surgeon to secure more satisfactory results. The very large number of fracture cases occurring in any community makes the problem important. This demand for better functional results in a shorter time is a just demand. How shall the demand be met?

My answer is, briefly, that the treatment of fractures may be improved:

I. By an organized fracture service in each of the large hospitals of the country.[1]

(*a*) Special wards should be used for men, women and children,

[1] One of the earliest fracture services in this country organized in a general hospital was gradually developed at the Massachusetts General Hospital, Boston, Mass.

and only fracture cases admitted. It is impossible to care adequately for these cases when they are scattered throughout the hospital. Responsibility is divided among many individuals. No concentrated interest results. Too much work is delegated to ignorant subordinates. The general service would be more free if separated from the fracture cases.

(*b*) A special fracture personnel should be in charge of these fracture wards. There should be a chief of this service; a surgeon of broad general experience, whose interest should be active in molding the policy of the fracture division. The chief should have absolute control of the policy of the division; he alone should be finally responsible for results. Serving with and under this chief should be the necessary assistant surgeons, resident house surgeons, and a nursing force. The service should be continuous throughout the year.

(*c*) This continuous control should include the out-patient service, where the ambulatory cases are received and treated. Each day of the week there should be an out-patient clinic for fractures, which the fracture service controls and with which it is in intimate touch. The policies of the out-patient and house fracture services should be identical under the chief of the service. Cases of fracture should be followed until the maximum functional results are obtained, and until the wage-earner is on his feet and reëstablished.

(*d*) The emergency ward or accident service, insofar as fractures are concerned, should likewise be under the direct care of the chief of the fracture service. A fracture received into the accident ward should be regarded as an emergency case requiring the immediate attention of those directly responsible for the ultimate result. A fracture should be considered as much an emergency as is a case of perforated gastric ulcer. The initial treatment is vital to a satisfactory outcome in both instances.

(*e*) An operation plant in connection with the house service is essential. The operative fractures must be kept apart from septic operations. Separate instruments must be employed.

(*f*) A lecture-room with easy access to the wards is necessary.

II. By adequate instruction of the undergraduate medical student.

The student should have a definite concept of a fracture of bone: What it looks like; what it is; the changes taking place in the tissues involved; the processes of repair. All these conditions should be accurately visualized by the student. Then the principles underlying the treatment of a fracture will be more intelligently grasped, based upon known and familiar anatomical, physiological and pathological facts. The undergraduate should be carried only a short distance in fracture instruction and only with illustrative cases.

Unfortunately, undergraduate medical students are overtaught today. The curriculum is crowded. The special branches should be taken from the routine curriculum and instruction limited more closely to general principles. This instruction in fractures should come in the third and fourth years. The medical school is the place to look for fundamental improvement in fracture treatment. Let us study to improve methods of elementary and graduate instruction in fractures. Good results are then bound to follow.

III. By instituting smaller hospital units in towns adjacent to and remote from large centers. An educational propaganda should be started which would result in a suitable fracture equipment being installed in each hospital. Such an equipment would afford opportunity for the instruction of certain interested physicians or surgeons of the community in the use of this equipment in actual cases of fracture. This educational propaganda might well be undertaken by individual surgeons of a large center as definite educational missionary work.

IV. By the graduate instruction of the general practitioner interested in fractures. The graduate medical school courses can readily supply this need. The details of treatment can here be covered very thoroughly.

V. Through the formal instruction of medical students intending to specialize in this branch of surgery. The infinite details of treatment should not be taught to undergraduates, but should

be taught the special undergraduate student in the graduate-school courses.

VI. By encouraging the specialization within general surgery of the surgery of fractures. Traumatic surgery today covers a complicated and varied field. I believe there is ample scope for such a specialty.

There will always be physicians in the community who will want to do everything; but there will also be some wishing to excel in a special field.

The establishing of a specialty of fracture surgery or of traumatic surgery in our larger centers should conduce to (*a*) better service to the community and (*b*) a more rapid advance in the knowledge of the treatment of special fractures.

At present, in a large hospital it may be more practical for the neurological surgeon to care for head injuries and spinal injuries; for the thoracic surgeon to care for the pneumothorax following penetrating lung fractures of the ribs; for the genito-urinary surgeon to repair a ruptured bladder after fracture of the pelvis; for the abdominal surgeon to take care of the ruptured intestine in a crush of lower rib or pelvis; but the chief of the fracture service should be a man so trained as to be able to handle any such complication that occurs in his division. Thus, the special fracture service or traumatic service would occupy a dignified position along with the other special services.

VII. By the organization of a clinical surgical fracture society, meeting once a year, for the sole discussion of fracture problems. The membership in such an organization should be carefully safeguarded.

Therefore, as Hey-Groves has put it, "By segregation of cases, continuity and teamwork," by the properly organized instruction of the young men of the medical schools of the country working through special fracture services, by educational propaganda among the smaller towns, by carefully arranged graduate-school instruction of medical practitioners, an influence may be gradually exerted which will eventually bring the results of fracture treatment to a high level, save dollars to the laboring

man, save time to industry, and further the scientific investigation of problems connected with bone repair.

One query has grown out of this World War which is a very important one and so widespread that it must be mentioned and answered. Orthopedic surgeons did splendid work during the late war. Only admiration is had for their early and complete organization; only praise for all they helped accomplish. Returning from the traumatic surgery of the war to civil life, the question is being forced upon surgical staffs of hospitals all over the the United States and Canada—Shall fractures of bone and traumatic surgery as outlined above be delegated in the first instance to members of the orthopedic staff of our hospitals?

General practitioners of medicine must, of course, always take care of fractures to a greater or less extent, especially in rural communities. Such men should have opportunities to become familiar with good methods, especially of first treatment afforded by graduate school courses. The general practitioner always has at hand some surgeon in a smaller or larger center who should be competent in this kind of case. To him he will defer doubtful matters about any case. Fractures, traumatic surgery, should always remain a part of general surgery. No one should assume the care of fractures who is not fitted to meet properly the very serious complications, sequelæ and attendant emergencies of fracture treatment.

Unfortunately, many good men are in orthopedic work who are not qualified as general surgeons. The opposition, I take it, that obtains on the part of the general surgeon to orthopedic men being assigned to the care of fractures rests solely upon the fact of their lack of general surgical experience. If in any community *competent* orthopedic men exist, of course they may properly be given the care of fractures.

My personal feeling is that the working out of this problem is to be a long and tedious one. We all want to see fractures and traumatic surgery better cared for. If the ground taken by the President of the American Orthopedic Association, in his presidential address this year, is realized, viz., "a most complete

early training in general surgery of all those who would become orthopedic surgeons," then an improvement will have been made in one direction. The door to any special surgical work should be always through general surgery. Unfortunately, many begin special work by short cuts, which may definitely leave the stamp of narrowness and inefficiency upon the individual and his work.

Let the valuable special orthopedic training be conserved for the fracture service. Let us not make the mistake of immediately placing orthopedic men (yet untrained surgeons) at the head of special fracture services. Let us rather make the competent orthopedic men consultants to such fracture services under the present leadership of a chief of service—a general surgeon acutely skilled and interested in fractures and traumatic surgery. We then will preserve the proper relationship between the specialty of traumatic surgery or fracture surgery and general surgery, and we shall gradually enrich this specialty by the brains of men interested in mechanical problems *per se*.

The time is coming when young men of this country will specialize in traumatic surgery. They will not be orthopedic surgeons doing traumatic surgery. They will be primarily surgeons of traumatism. The present scope of orthopedic surgery is sufficiently large to satisfactorily occupy the orthopedic surgeon. There are ample reasons for a definite specialty in traumatic surgery as outlined above.

THE MECHANICS OF PRODUCTION AND THE TREATMENT OF SPIRAL FRACTURES

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The problem presented by spiral fractures of the long bones is one of special importance, first because of the great frequency of these fractures and second because the anatomic and functional results of their management by the usual technic leave much to be desired.

Figures differ as to the relative frequency of spiral fractures because of more or less uncertainty as to the definition, *i. e.*, in addition to pure torsion fractures there are mixed forms in which torsion may be active in the production of the fracture but plays a relatively small part, and again there are opportunities for difference of opinion as to what proportion of the so-called oblique fractures may properly be classified as spiral fractures.

For purposes of the present paper only those fractures are called spiral which are typical and are the result of torsion. Stimson (edition 1912) says spiral fractures are rare. Scudder (1911) does not mention them. Zuppinger estimates that 26 per cent of all fractures of the tibia are spiral and 39 per cent of all fractures of the shaft of the tibia. Probably these figures are low.

That the results of the treatment of spiral fractures by the process of reduction and fixation, and even by traction, are unsatisfactory anatomically and functionally may easily be seen, and will as readily be admitted by almost any clinician

who will carefully criticise and check up his results. Non-union is frequent in spiral fractures of the lower third of the tibia; irregularity of the bone in case of union is the rule with a persistent and symptom-producing external rotatory deformity in the majority of cases, with frequent anterior flexion deformity and more or less shortening. We often see abduction deformity as well, sometimes of sufficient degree, especially when associated with external rotation, to be the cause of breaking down of the arch of the foot with resulting pronation and flat-foot.

A proper understanding of spiral fractures and the special difficulties of their management must rest on a clear conception of the mechanism of their production, and fortunately this is not difficult, though it receives scant attention even in our better texts.

That spiral fractures of the shafts of the long bones are the result of torsion has long been known and abundantly proven, both clinically and experimentally—witness the unusually interesting case of Dr. Monks of this Association—in which no force other than torsion was active. In this case two men were sitting with forearms flexed to a right angle, elbows on a table between them, striving by pressing their palms together to see which could force the other's hand to the table, thus producing powerful external rotatory stress on each humerus. One humerus gave way, breaking in a spiral. Precisely the same thing occurred in a personal case in which the right humerus gave way in a left-handed spiral fracture as a result of violent effort in throwing a base-ball.

If a fracture is produced by a mechanism so simple that it can be followed and analyzed one would expect the form of the fracture to be typical under the laws of pure mechanics. These conditions prevail most perfectly in the case of spiral fractures where not only the general form of the fracture but also the direction of the spiral is determined by and corresponds to the direction of the axial rotation. Moreover, most of the clinical phenomena presented by spiral fractures are such as necessarily must obtain as a consequence of these mechanical relations.

The fundamental principle determining the direction of the spiral and the pitch were worked out by Zuppinger,[1] applying Hooke's law to the effect that the lengthening of a given bar of any material under tensile stress is proportional to its original length and to the tensile force active in producing the lengthening. If a bar of any cross-section, *e. g.*, a cylinder of length h is subjected to axial torsion, and if L be the length of any line drawn *ad libitum* on its surface the effect of the torsion is to move the point A to some point A' in the direction of the torsion, l would then be the amount of lengthening of L resulting. (Fig 1.)

The following equation would represent the relations:

$$S = a \frac{l}{L}$$

in which S is the tensile force and a some constant depending on the nature of the material. By simple algebraic substitution it is evident that

$$S = a \frac{p}{h} \sin \varphi \cos \varphi$$

for small values of p. S is a maximum where sine and cosine are equal, *i. e.*, at $\varphi = 45°$.

From this it follows that the more brittle the material the nearer the angle approaches 45 degrees. Thus if one twists axially a chalk crayon, a material approaching the limit of brittleness, he finds that it breaks consistently in a spiral of almost exactly 45 degrees' pitch.

The material in giving way breaks at right angles to the direction of the tensile force. Therefore a bar subjected to left-handed torsion, as in the figure, breaks in a left-handed spiral, and, *vice versa*, right-handed torsion causes right-handed spiral fracture. Since in less brittle material there is a greater yielding or stretching under tensile stress (in this case torsion) the angle (φ) is small, consequently the pitch of the spiral fracture is steeper. Thus, in the long bones of children spiral fractures are much more oblique than in adults, which accords with

[1] Beitr, z. klin. Chir., 1906, lii, 391 and 1909, lxiv, 562.

clinical experience—extreme cases occurring in which the spiral fractures are almost longitudinal fractures. It thus follows that in children many spiral fractures are capable of practical adjustment without open operation.

The effect of torsion may readily be illustrated by cutting a piece of rubber tubing in a spiral manner and then twisting it axially—a right-handed spiral will be opened up by a right-handed torsion, and, *per contra*, a left-handed spiral will be opened up by a left-handed twist.

The almost universal finding clinically of left-handed spirals in the right upper and lower extremities and right-handed spiral fractures in the left upper and lower extremities is the result not of any particular characteristic of constitution of the bones in question but purely of the mechanical necessities of the case, a measure of the overwhelmingly greater frequency with which the extremities are subjected to external rotation rather than internal.

It is but a coincidence that in the development of the extremities a rotation occurs in the long bones in the sense of internal rotation, which is supposed by some to produce a sort of spiral grain in the bones (Flemming). Witness the backward position of the feet in the so-called siren deformity where internal rotation is inhibited by fusion of the fibulæ. Here, the spiral development of the femora continuing, the upper end of each femur gives way in an anterior congenital dislocation of the hip (Dreesmann).

If a right femur or humerus be subjected to internal rotatory stress sufficient to break it, *e. g.*, right-handed torsion, a right-handed spiral fracture will result in spite of any left-handed spiral grain to the contrary, and this right-handed spiral fracture will be identical, except for the reverse direction of its spiral, with the usual left-handed spiral fracture of the right femur or humerus.

The spiral fissure may extend completely around the full circumference of the bone even more than once. In the case of the young man who broke his humerus throwing a base-ball

the spiral fissure when exposed in open operation was traced at least twice around the bone.

Since in a given case of torsion effective in breaking a long bone the whole bone is under stress in an infinitude of potential spirals, determination of the particular spiral in which the bone is about to break is quite fortuitous, depending simply on the location of some weakest point. Of course this is ordinarily single, but it is not a very violent supposition to conceive that there may be two or more weakest points so nearly equal that the bone may break in two or more parallel spirals. With this idea in view, and after hunting for some years, I finally found a case of spiral fracture of the tibia with parallel spiral fissure in a young adult. Of course, others have found similar cases. Recently my associate, Dr. Rumwell, found one with two parallel spiral fissures in a girl, aged twelve years, in the Stanford outpatient clinic, and Dr. Blaisdell, of the Stanford Medical School, succeeded in producing by pure torsion parallel spiral fractures in the humerus of a cadaver.

If the torsion force acts no further the bone is merely spirally fissured; it is not actually separated into two fragments. Precisely this fracture does occur occasionally in young people, as is proved by the *x*-ray.

It is not necessary to invoke other forces, such as bending moment, as does Zuppinger, to explain the occurrence of the vertical component of spiral fractures for this part of the fracture, by which the bone is separated into two fragments, necessarily follows from continuance of the torsion.

That bending moment, however, has some effect in determining the particular location of the spiral component of the fracture is probable as offering an explanation (Zuppinger) of the fact that in the majority of instances of spiral fracture of the tibia the spiral component is ordinarily on the anterior aspect of the bone and the longitudinal component behind.

If the torsion force continue after the bone has given way in spiral fissure an entirely different distribution of forces instantly takes place. Tensile forces develop on the inner surface of the

bone and compressive on the outer, as may readily be seen by reference to the diagram. (Fig. 2). The bone tends to open out

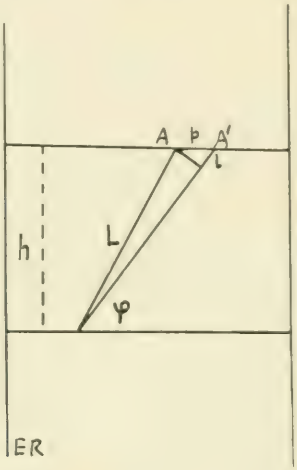

FIG. 1.—After Zuppinger.

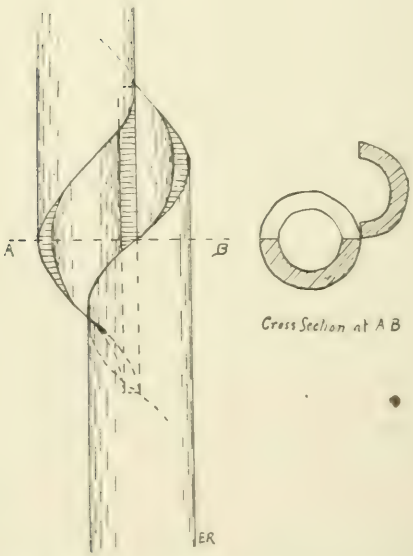

FIG. 2.—Right handed spiral fracture with fragments rotated to 90

into a flat ribbon, and presently, giving way to the tensile forces now active on its inner surface, it breaks in an early longitudinal fracture, folding outward on its external surface and on the periosteum as a hinge. This longitudinal fracture connects two adjacent limbs of the spiral, completing the separation of the bone into two fragments. Since the spiral fracture precedes the longitudinal the spiral fissure is ordinarily found to extend a considerable distance at one or both ends beyond the point of intersection of the longitudinal element of the fracture.

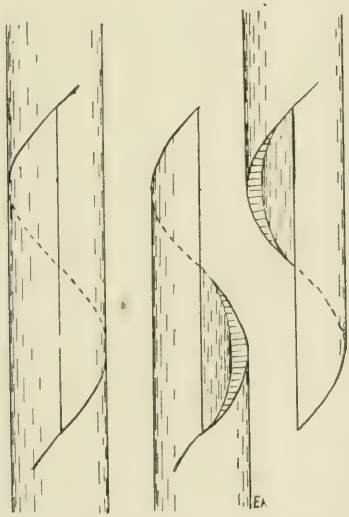

FIG. 3.—Right-handed spiral fracture seen from side of longitudinal component.

Since the bone is pulled apart along the spiral element of the fracture the periosteum is uniformly torn along this line, and because in the production of the longitudinal component of the fracture the bone is folded toward its outer surface the periosteum is not torn along this part of the fracture. This fact is of the greatest clinical significance, as will be seen farther on.

Along the spiral part of the fracture, however, the periosteum,

as said above, is torn; more than that, it is pulled apart and therefore torn irregularly, leaving numerous tabs which fold around the fracture surfaces, and if not removed or replaced militate against union much as similar tabs of aponeurosis do in transverse fracture of the patella.

By reference to the diagrams again it may readily be seen that in the process of disengaging the tips of the fragments in the instant of completion of the fracture, their sharp points being accurately mortised in their corresponding niches, will frequently be broken off because the majority of long bones are of substantial thickness between periosteum and marrow, varying, of course, but averaging perhaps one-fifth of the diameter of the bone.

So much for the effects of torsion alone. In practice we find that other forces are active in many cases at the moment of fracture and afterward. Especially is this true in the lower extremities where weight-bearing, augmented in its effect by momentum in running or jumping, adds an important element of longitudinal thrust. While it would be interesting mathematically to estimate the effect of such longitudinal thrust in increasing the pitch of the spiral by adding shearing stress, this effect is unimportant clinically. Much more important is the effect of longitudinal thrust if active after fracture is complete and the fragments rotated as described above; for by this same rotation the fragments are completely disengaged and offer no resistance whatever to longitudinal thrust. Thrust will then cause the fragments to pass by each other, and the periosteum on the side of the vertical component of the fracture not being torn apart will be stripped from one or both pointed ends of the fragments but otherwise will remain as a "periosteal bridge." Clinically, one generally finds the tips of the fragments in spiral fractures bereft of periosteum.

The fragments under these circumstances are so free to move in any direction that their sharp, projecting points and knife-like edges may lacerate muscles, nerves, bloodvessels, neighboring joints, etc., and often perforate the skin, making the

fracture compound. In being moved about these long, sharp points often catch between them tough fibrous tissues, such as intermuscular septa as well as nerves and bloodvessels, any of which may effectively prevent reduction of the fracture.

Moreover the long points of the fragments may be broken off by bending the limb at the point of fracture, and this the more easily occurs because the long points are weakened by the fact that the spiral fissures often extend far beyond their intersections with the longitudinal component of the fracture.

From the above considerations and the complicated form of the spiral fracture it is evident that if the fracture is not perfectly reduced there is practically no reduction at all; *i. e.*, if there is any displacement whatever, there is a great deal of displacement. The untorn "periosteal bridge," being attached along the sides of the bone more or less opposite to the spiral component of the fracture, is short, and it is made still shorter by the deposition beneath it of chips of bone or blood-clot, or, if a little time has elapsed since the occurrence of the fracture, by inflammatory tissue or new bone. This short "periosteal bridge" effectually prevents overcoming of rotatory displacement, and is one of the reasons for the common clinical experience of the persistence of external rotation after union of a spiral fracture.

From a practical standpoint spiral fractures of the long bones are never reduced except in open operation. But it may be argued that by traction and splinting shortening may be overcome and the fragments may be laid alongside each other, overlapping by their long tongue-like processes, affording sufficient contact to secure bony union. It may readily be seen from the diagrams that unless the fracture is accurately reduced it gapes widely along the spiral component, and where contact is established the raw edge of the bone is to a considerable extent in contact with the outer surface of the torn but not stripped-up periosteum. A dead space is there present, lined on one side by connective tissue (outer surface of the periosteum), which may grow into it more rapidly than bone, militating strongly toward non-union.

Thus may readily be grasped the reason for the common clinical experience that union begins on the side of the "periosteal bridge," *i. e.*, on the side of the longitudinal component of the fracture, the intact periosteum furnishing an effective barrier to the ingrowth of connective tissue at this point.

On the other hand when the fracture is accurately reduced the fragments fit with such mathematical accuracy that they afford a very considerable degree of stability. Theoretically in the typical case there is but one direction in which stability is lacking, and that is in the direction of the original axial rotation by which the fracture was produced.

It would therefore be logical in the management of such fractures to support this weak point. Like accurate reduction of the fracture this can only be accomplished in open operation, but then it can be done most simply and effectively with a minimum of traumatism to the tissues and with a minimum of implanted hardware. There is no need for long plates with many screws.

By giving scrupulous attention to the principle of producing a minimum of traumatism, avoiding stripping up of the periosteum and with a good aseptic technic, the risk of infection would be minimized, and it would seem that the advantages of securing perfect form and function otherwise impossible would warrant taking that risk. If operation is done early, *i. e.*, before secondary changes have occurred, the required manipulation is easy and there is no necessity of excessive pulling and prying and tearing the tissues.

While not an advocate of operative treatment in fractures in general, or even in any very large proportion of cases, I submit that in my hands I have had far greater satisfaction in view of the results obtained in early operative reduction and fixation of spiral fractures of the long bones than in treatment by traction and external fixation. It is not fair to the treatment of fractures by open operation to have operation relegated to the position of last resort. It should be possible in most fractures to determine before treatment is begun whether the chances for

union without deformity are better with or without open operation. There should be no such thing as operating for malunion, for it can always be determined in the first few days of treatment of a fracture case whether proper position can be maintained before embarrassing secondary changes have occurred.

The rapid degeneration of bone under non-use readily appreciated by following the increase of transparency to the *x*-rays militates against later union. It is for this reason and for the maintenance of joint integrity that massage of muscles and mobilization of joints is so important in the treatment of fractures. Of greater value than massage and passive motion in maintaining circulation and nutrition of the injured limb is functional demand, a fact that has long been evident from a clinical standpoint, though not always carried out in practice as systematically as it should be. For the experimental determination of the value of functional demand on the growth of bone I would refer to the beautiful experiments on the bones of young dogs by Dr. Haas, of San Francisco, in which the beneficial effect of early resumption of function is shown in contrast with the findings in an ample series of control experiments.[1]

If fractures of the long bones can be so adjusted and held as to permit early functional demand, non-union will have lost one of its chief causes. In transverse fractures if the fragments can be set end on and held so even if the area of contact is but a fraction of their surfaces the arm and forearm can be used a great deal and in the lower extremity weight-bearing may early be established. In spiral fractures if accurately adjusted in early operation and the weak part of the mechanism properly supported a considerable functional demand can be made within a comparatively short time.

In view of the foregoing it would seem to be worth while to attempt to establish a definite technic for the management of spiral fractures, at least those in which the mechanical phe-

[1] To be printed in the Archives of Surgery.

nomena can accurately be estimated, and a technic ought not to be difficult to find on which surgeons could agree.

In spiral fractures of the long bones the advantages of the early operative treatment are so great that I would submit that, barring definite contraindications to operation in general, all spiral fractures of the long bones in adults and adolescents, and some in children, be managed by open early operation, and would submit the following as a technic which has given excellent results in my hands:

1. Determine the location and form of the fracture by *x*-ray plates so taken as to locate that part of the spiral portion of the fracture which is opposite the longitudinal component.

2. Cut down on this spiral part of the fracture and remove all detached chips of bone and larger fragments if not required as a part of the splintage and drill both fragments, if possible, *in situ* or with a minimum of disturbance of their position, locating the holes at such points as after reduction will make the line joining them lie obliquely to the spiral in suchwise as most effectively to resist torsion displacement. In general this line will be transverse to the axis of the bone.

3. Pass a stout silver wire through the drill holes.

4. Reduce the fracture by traction, rotation and leverage, taking care not to break the bone, lifting out any periosteal or fibrous tabs from between the fragments along the spiral, but taking care not to strip up the periosteum. Draw the wire taut and twist, hammering the ends down against the bone.

5. Close the wound and apply some sort of efficient retentive appliance, a properly fitting Thomas splint or plaster-of-Paris cast.

6. Remove the retentive appliance frequently for massage, mobilization of the joints and electric development of the muscles and arrange for the patient to make functional demand at the earliest reasonable moment.

THE VALUES OF THE VARIOUS METHODS OF BONE GRAFTING, JUDGED BY 1390 REPORTED CASES

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Surgeons have been experimenting with bone graftings, and a sufficient number of the various methods have now been performed to form a basis of their comparative values. All graftings reported in the literature,[1] together with the results obtained from a questionnaire, have been analyzed. In these statistics, methods are considered successful only when grafts have produced a satisfactory amount of new bone. Valuable conclusions can only be drawn from the results obtained by the employment of human bone, experiments in dogs being of little or no value in demonstrating either the best method of grafting or the reasons for the success or failure of the method employed. In dogs, it is impossible to produce non-union of a fracture except by interposing soft tissues. In the human, non-union of a fracture frequently occurs, and while many are due to faulty treatment, a considerable number cannot be explained on any known hypothesis.

It seems true, with the possible exception of a few cells on the surface of a bone graft which are not cut off from their blood supply, that all the adult bone cells in the interior of a bony transplant necessarily die because of their being deprived of their nutrient supply—that is, they never proliferate a new bone cell. If this be correct, the objection to the use of the electric motor saw, on the ground that the generated heat kills the bone cells, has no weight. The practical results of bone

[1] Excepting Albee's statistics which were not available.

graftings show that there is little difference in the final success or failure of a bone graft whether it is made by a motor-saw or chisel. Too much importance has been placed upon the bone cell as the essential element in bone regeneration, and this error has obscured a right conception of the process. Bancroft says: "Bone is mesoblastic in origin, and in its repair we find that calcium salts are deposited on the intracellular elements of connective tissue, forming new bone. The connective-tissue cell, then, by a process of metaplasia, becomes a bone cell. Periosteum is a connective tissue and hence is prone to form bone, but it is not the *only* connective tissue that has this function." Neuhoff has performed some interesting as well as astonishing experiments on dogs. He transplanted fascia lata into defects made in the bladder, ureteral and stomach walls, to see how connective tissue would act when introduced into these localities. To his astonishment bone was formed in the fascial (*i. e.*, connective tissue) transplants in each of these three positions, that is, in a position where the transplants were each bathed in an acid fluid.

Since the tendency to the formation of connective tissue is universally present in the tissues, it seems reasonable to argue that there must be some peculiar chemical influence present in a particular location to cause the precipitation or deposition of calcium in connective tissue in order to start the formation of bone. That acidity is not necessarily the determining factor in new bone formation seems to be demonstrated in the formation of new bone in extraskeletal tissues, such as arteries, ovaries, kidneys, myositis ossificans, etc., where the alkaline blood bathes the tissues. Some chemical combination in the blood in association with some local stimulus, rather than the reaction of the blood, seems necessary to form new bone in connective tissue. Perhaps some day the physiological chemist will discover some substance to inject into connective tissue which will precipitate calcium and start the process of forming new bone.

Connective tissue seems to be essential in the formation of bone. Osteoblasts are indistinguishable from fibroblasts. The

first occurrence in bone formation is the arranging of fibroblasts (osteoblasts) around a bloodvessel. In this new fibrous tissue, calcium is deposited by some unknown influence, which goes on to the formation of bone. If calcium is deficient, fibrous union results, due to some chemical deficiency. If there is too much mobility between the fragments, there is a stage of cartilage formation which *may* eventually become changed into bone. Where there is no mobility, as in the fracture of one bone with a parallel bone acting as a splint, there is no formation of cartilage.

There are three requirements of a successful bone graft:

1. It must bridge a defect.
2. It must be of a size and type to reëstablish the circulation.
3. It must act as a stimulus to osteogenesis. Raw living bone is a very powerful stimulus to osteogenesis. The osteoperiosteal method offers a very large area of raw bone, hence is to be preferred to all other methods of grafting.

There is much in osteogenesis that is still unknown, namely, the chemistry and the physiology of the process. Why is it that sometimes a bone graft which has remained in place without suppuration will gradually melt away in the tissues, will become gradually absorbed, and its place not be taken by new bone? This is one of the most disappointing results of a well-conceived and well-carried-out bone-grafting procedure that can happen, and it has occurred to all of us. It makes no difference what method is employed in grafting, it occurs in all methods, whether the periosteum is on the graft or not.

Table I shows a total of 1390 patients upon whom bone graftings were performed, and of these 1145, or 82.3 per cent, succeeded, while 245, or 17.6 per cent, failed. Successes (82 per cent) were the same proportionately whether periosteum was on the graft or not. Analysis of the various methods shows that 95.8 per cent of bone pegs were successful, these being, of course, without periosteum or endosteum upon the grafts. The osteoperiosteal method, developed by the French school (particularly Delageniere), proved the next most successful. This method consists in molding about the fragments strips of periosteum,

taken from the tibia, to which adhere thin plaques of bone. Successes by this method were 87.3 per cent. By the end-to-end without inlaying method, favored by the English surgeons, 82.5 per cent of successes were obtained, and by the inlay method, most in favor in America, 80.9 per cent were successful. By the intramedullary method, developed by Murphy, only 76.6 per cent of successes resulted. The intramedullary graft at one end, combined with an inlay at the other, with 60 per cent of successes, proved the least successful of all, possibly because of the liability to dislocation of the graft. This would tend to show that the intramedullary method should be discarded as a method of bone grafting.

TABLE I.—RESULTS OF REPORTED METHODS OF BONE GRAFTING.

COMBINED STATISTICS.

Total number of cases, 1390: Successes, 1145, or 82.3 per cent. Failures, 245, or 17.6 per cent.

With periosteum, 1170: Successes, 970, or 82.9 per cent. Failures, 200, or 17.1 per cent.

Without periosteum, 196: Successes, 162, or 82.6 per cent. Failures, 34, or 17.4 per cent.

Suppuration in 121 cases, or 8 per cent: Successes, 32 per cent. Failures, 68 per cent.

TABLE II.—METHODS

1. Bone pegs, 24: Successes, 23, or 95.8 per cent. Failures, 1, or 4.2 per cent.

2. Osteoperiosteal, 426 (Ollier, Codivilla, Delageniere): Successes, 372, or 87.3 per cent. Failures, 54, or 12.7 per cent.

3. End-to-end (without inlaying), 166: Successes, 137, or 82.5 per cent. Failures, 29, or 17.5 per cent.

With periosteum, 74: Successes, 54, or 72.9 per cent. Failures, 20, or 27.1 per cent.

Without periosteum, 92: Successes, 83, or 90.2 per cent. Failures, 9, or 9.8 per cent.

4. Inlay, 540: Successes, 437, or 80.9 per cent. Failures, 103, or 19.1 per cent.

With periosteum, 494: Successes, 396, or 80.1 per cent. Failures, 98, or 19.9 per cent.

Without periosteum, 46: Successes, 41, or 89.1 per cent. Failures, 5, or 10.9 per cent.

5. Intramedullary (Murphy), 214: Successes, 164, or 76.6 per cent. Failures, 50, or 23.3 per cent.

With periosteum, 140: Successes, 117 or 83.5 per cent. Failures, 23, or 16.5 per cent.

Without periosteum, 64: Successes, 45, or 70.3 per cent. Failures, 19, or 29.7 per cent.

6. Combined intramedullary (one end) with inlay (other end), 20: Successes, 12, or 60 per cent. Failures, 8, or 40 per cent.

Further analysis of the effect the presence or absence of the periosteum has upon the results of the various methods of bone-grafting shown in (Table II):

3. *End-to-end.* With periosteum: Successes, 73 per cent. Without periosteum: Successes, 90 per cent.

4. *Inlay.* With periosteum: Successes, 80 per cent. Without periosteum: Successes, 89 per cent.

5. *Intramedullary.* With periosteum: Successes, 83.5 per cent. Without periosteum: Successes, 70.3 per cent.

Thus, with the end-to-end method there were 17 per cent fewer successes, and with the inlay there were 9 per cent. fewer successes with grafts *with* periosteum than *without;* while with the intramedullary method there were 13 per cent *more* successes with grafts *with* periosteum than without. So that we must draw the conclusion that, in the end-to-end and inlay methods, periosteum on the grafts seems to be slightly disadvantageous, while on the intramedullary grafts it acts more advantageously *with* than without.

Ashhurst has reported 27 inlay bone graftings without periosteum with 22 successes (81.5 per cent), and Gallie 18 inlays without periosteum with 100 per cent of successes.

It is difficult from these statistics to measure accurately the value of periosteum on grafts. Theoretically, most surgeons believe it of value; though not considered essential, it is supposed to assist in establishing a new and richer circulation, upon the extent of which the amount of the formation of new bone seems to depend. Several reasons may be advanced to account

for the relatively poor results of intramedullary grafts: (1) Because of the obliteration of the nutrient artery, from which the graft largely obtains its blood supply, through thrombosis, antecedent trauma or operative procedure; (2) because of the liability of dislocation of the graft; (3) because of its susceptibility to fracture; (4) because of the difficulty of adequately immobilizing, or anchoring the graft; (5) because, in the opinion of some, of the lack of periosteum. In case of suppuration the success of the transplantation has been deemed more likely *with* periosteum than *without*, for the periosteum is much more resistant to infection than bone. In illustration of this fact, I have reported[2] one case of an inlay graft with periosteum, made for a defect in the lower jaw, which suppurated, losing by sequestration almost its entire bone content, which eventually proved completely successful through the reformation of the bony segment, which presumably came from the transplanted periosteum.

Fisher[3] says: "From a study of the pathological appearances in the cases of loose bodies of bone and cartilage quite free in the joint, it is clear that the majority of the bone corpuscles were dead, as exemplified by the lacunæ no longer holding stainable cells. Furthermore, in not a single case was there any evidence of proliferation of osteoblasts with formation of new bone. Yet we see a striking difference on examining those bodies of traumatic origin and containing bone which have acquired an early adhesion to the synovial membrane; for, in these, fresh capillaries can be seen to have penetrated the cancellous spaces and active proliferation of osteoblasts with formation of new bone is taking place. Exactly the same thing can be observed in the experimental production of loose bodies, which show secondary attachment to the synovial membrane with active proliferation of bone cells. These observations, therefore, support Hey Groves' contention that the osteogenic cells in the Haversian canals and cancellous spaces of an autogenous bone graft actively proliferate only if the graft becomes to some extent vascularized by the

[2] Ann. Surg., 1921.

[3] Lancet, April 23, 1921, p. 844.

ingrowth of capillaries from surrounding parts. As, therefore, the vascular supply seems to be of such paramount importance in the osteogenic function of the bone graft, it would appear to be advantageous to retain the periosteum in order to enable the graft to establish its vascular connections as rapidly as possible."

The part played by periosteum has been considered by Leriche and Policard: It seems to them that periosteum has two elements with antagonistic tendencies, but the juxtaposition of these two elements produces harmony in the process of osteogenesis. The element of bone growth is in the cambium layer of the periosteum (*i. e.*, the juxta-osseous connective tissue and the adjacent bone) and that which arrests bone formation is the outer fibrous layer of the periosteum. Between the two there is normally a state of equilibrium and leads to the entire series of osteogenesis, hence the periosteum is the whole manageable agent of surgical osteogenesis. By the stripping up of the periosteum the cortical layer of the bone regains its power of extension, new bone appears —pushed forward to establish a new state of equilibrium. This view seems to be substantiated by the results of subperiosteal resection of bony prominences, in which new bone is formed beneath the periosteum, not outside of it, unless the periosteum has been ruptured. Renfrew White says: "The osteogenic property of the periosteum (which it seems to possess) is in fact not its own, but due to a resumption of osteoblastic activity of the cells of minute portions of bone that have adhered to it, having been detached with it."

Sebileau[4] showed a specimen removed at autopsy ten months after an osteoperiosteal graft, four inches long, taken from the tibia, which had been transplanted to bridge a defect in the lower jaw. There was a firm, osseous callus between the two stumps, although the outer periosteal surface had been turned, being directly applied to the bone surfaces, reversing the ordinary procedure. This seems to show that a graft is endowed with vitality in itself.

[4] Presse méd., August 1, 1918.

A study of these results of the various methods of bone grafting shows that the method most likely to achieve success is the osteoperiosteal. That this method is applicable in large defects is shown in a case reported by Katzenstein,[5] where 13 cm. of the diaphysis of the femur were missing. In the end of each fragment he hollowed out a cavity which received the extremities of the transposed, periosteal strip, to which were clinging thin plaques of bone of the thickness of a dime. Immobilization was maintained for six months, when the defect was filled in with new bone and the man was able to walk without support. An advantageous amplification of this method would be, after removing the periosteal strips from the tibia, to take bone chips from the raw tibial surface with a gouge and to implant these chips so as to fill in the defect (Macewen) between the ends of the fragments.

The osteoperiosteal flaps overlap the ends of the fragments to which they are sutured with catgut. The advantages of this method include:

1. Increased liability to success of the grafting.

2. Greater simplicity of the technic of obtaining the graft, there being no necessity of complicated and expensive motor saws.

3. Decrease in the liability of subsequent fracturing of the tibia from which the graft is taken, of which there are numerous reports in the literature.

Macewen has recorded successes after implanting small bone chips in the human, and Bancroft has had successes with the same method. A much better method than to fill a bone cavity with fat after sterilization is to fill the cavity with a curled-up osteoperiosteal flap. By this procedure the cavity becomes filled with new bone.

CAUSES OF FAILURES OF BONE GRAFTINGS

1. *Improper Method Employed.* From Table II it is seen that the osteoperiosteal method is attended with the greatest number

[5] Rev. de chir., May, 1910, p. 952.

of successes and the intramedullary method with the smallest number. The presence or absence of periosteum seems to be immaterial

2. *Suppuration.* This is apparently the most evident cause of failures in bone graftings. Walker[6] reports that in 62 per cent of 46 cases in the army (Surgeon-General's statistics) which suppurated after graftings the transplants were finally successful. These results are clearly too favorable, because in the author's statistics of 1390 graftings, successes were 82 per cent; hence Walker's statistics show an increase total of only 20 per cent injuriously affected by suppuration. Of the author's reported 121 cases (8 per cent of all) which suppurated, only 32 per cent were successful.

3. *Immobilization.* Insufficient immobilization, or over too short a period, seems to be a very patent and frequent cause of failures. The most perfect immobilization that can be obtained after graftings should be employed. Brooks has shown that the absorption of bone in a bone graft is not complete before the end of two months. In addition, regeneration of bone as a result of a bone graft depends primarily upon the blood supply to the graft. These new-formed bloodvessels are very easily ruptured by mobility of the fragments, thus compromising the necessary blood supply to the graft. Hence the suspension method of treating bone grafts in a Balkan frame would seem to be contra-indicated, as allowing too much mobility between the fragments and the graft. Massage is likewise contra-indicated for the same reason. From four to six months' absolute immobilization is the minimum time in which it should be employed, unless careful testing shows consolidation to be complete before this time.

4. *Fracture and Dislocation of the Grafts.* These likewise are often the cause of failures and are frequently due to insufficient immobilization.

[6] Ann. Surg., January, 1921, p. 2.

5. *Atrophy of the Ends of the Bone Fragments.* No grafting should be undertaken if a radiogram shows the ends of the bones to be markedly atrophied, which is caused by deficient nutrition from too tight or too long prolonged immobilization, suppuration, neurotrophic disturbances, senility, etc. In such cases measures should be instituted before grafting is performed to stimulate the function and the circulation of the parts, by removing all splints, allowing free use of limb, Bier's hyperemia, bakings, massage, etc.

6. There is no evident explanation of some failures of bone graftings. Despite aseptic healing-in of the grafts a certain proportion of them (17.6 per cent) will fail, indicating some chemical or physiological deficiency of which we are as yet ignorant. Greater efforts, in future, must be made along chemical and physiological grounds to discover the elements which initiate new bone formation, as well as those which continue the process when once it has started.

Conclusions.

From 1390 bone graftings, we find:

1. That there was a total of 82.3 per cent of successes, with 17.6 per cent of failures.

2. In the order of successes, we have—

   (*a*) With bone pegs, 95.8 per cent were successful.

   (*b*) With the osteoperiosteal method (Delageniere), 87.3 per cent were successful.

   (*c*) With the end-to-end method (without inlaying), 82.5 per cent were successful.

   (*d*) With the inlay method, 80.9 per cent were successful.

   (*e*) With the intramedullary method (Murphy), 75.6 per cent were successful.

   (*f*) With the combined intramedullary (at one end) and the inlay (at the other), 60 per cent were successful.

3. The presence or absence of periosteum seems to exert no

influence on the success of bone grafts. Proportionately, the percentage of successes without periosteum (82.3 per cent) is the same as with (82.9 per cent). In the end-to-end method there were 18 per cent more successes than failures without periosteum, and in the inlay method 9 per cent more successes without periosteum than with; while, on the contrary, with the intramedullary method there were 13 per cent more successes with grafts *with* periosteum than without. It is difficult to explain the cause of the differences in the various methods.

4. Suppuration occurred in 121 cases, or 8 per cent; 32 per cent of these succeeded. Suppuration is the most frequent cause of non-success of graftings, with insufficient immobilization and too short duration as the second most frequent cause.

5. The conclusion is reached that the most successful method of bone grafting is by the osteoperiosteal method (Delageniere). The bony defect should be filled in with small bone chips, and on one or two aspects, overlapping the ends of the fragments covering in the bone chips, should be placed one or two strips of periosteum with adherent osseous plaques taken from another bone. This method is as applicable to large as to small bony defects.

6. The cause of many non-successes is due to defective immobilization or to undue curtailment of its length. From four to six months' immobilization is ordinarily required for complete success.

7. There is sufficient evidence to prove that the most effectual treatment of non-union of fractures is bone grafting.

8. The causes of failures of bone graftings, summarized, are—

(*a*) Improper method of grafting.

(*b*) Suppuration.

(*c*) Insufficient immobilization, or over too short a period of time.

(*d*) Fracture and dislocation of the grafts.

(*e*) Atrophy of the ends of the bone to be grafted.

9. The intramedullary method of grafting should be discarded.

THE SUSPENSION TRACTION TREATMENT OF FRACTURES OF THE LONG BONES NEAR LARGE JOINTS

By JOHN A. HARTWELL, M.D.
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The underlying principles which make for efficiency in the suspension traction treatment of fractures of the long bones are now well understood. Foremost among them is that this form of treatment takes into full consideration the necessity of caring for the damaged soft parts. Full recognition is given to the truth that in many instances muscular, fascial and joint repair are far more important in restoring a properly functioning limb than absolute anatomic restitution of the bone. This form of treatment also accepts the truth of the teaching of Lucas-Championnier, that complete fixation of fragments is not necessary for proper callous formation, in fact, may be detrimental to it.

In addition to these, the traction suspension treatment affords the best means of placing the distal fragment in proper alignment with the proximal fragment. In every fracture of a long bone, two elements enter into the resulting deformity. Either a proximal fragment is drawn from the planes of anatomic rest, while the distal fragment assumes such position, or the distal fragment is drawn out of the planes of the normally placed proximal fragment. Not infrequently, these two elements are combined. As a rule, the first effect is more marked when the fracture line lies near the proximal end of the bone, while the second is more prominent when the fracture line lies near the

distal end. Almost universally, the surgeon has better control over the distal than the proximal fragment. Therefore, treatment aims so to place the traction suspension forces that the former is drawn into proper alignment with the latter. Occasionally, it is helpful to supplement this with traction applied to the distal end of the proximal fragment. Our means for doing this are, as yet, not over adequate; but I am inclined to believe the method of Heitz-Boyer can be made to find a wider application than it has enjoyed.

It is my belief, that with proper attention, practically every fracture through the middle third of a long bone can be successfully treated by the traction suspension method, and that the results will be more promptly obtained and better functionally than by other methods. In other words, I have found the principles of this method sound and their application possible with this type of fracture. It must be emphasized, however, that the application is often beset with difficulties, and that patient, untiring effort will alone be rewarded with success. It is a method that can be applied only after considerable experience, and with attention to detail that many surgeons are unwilling to give. Its success, therefore, depends upon the opportunity to see and treat a considerable number of cases, coupled with a recognition of the fact that the expenditure of time and care will bring a satisfying reward. The hospital service—Cornell Division of Bellevue Hospital—where our study has been carried on, furnishes in abundance the former, and has been fortunate in having on its staff two men (Dr. Kenneth Bulkley and Dr. James Worcester) who have furnished the latter, and by their enthusiasm, inculcated their confrères with the desire to continue the work.

As a result of our experience, I repeat that only very exceptionally do we encounter a fracture in the middle third of a long bone which cannot be successfully treated by the traction method. Every fracture is made an emergency case in that its treatment is immediately undertaken on admission. A preliminary roentgenogram is taken.

The method of applying traction in fractures of the femur is by means of the ice-tongs, as modified by Dr. Worcester (Fig. 1), and suspension is obtained by the Thomas splint and the Balkan frame (Fig. 3). In an adult male, the initial weight applied is twenty-five pounds or more. This is reduced when the roentgenogram indicates that it is possible. It cannot be overemphasized that the immediate application of sufficient weight to overcome the shortening is all important. Over pull is preferable to under pull. Too little weight at first results in a fixation of the injured muscles in a

FIG. 1.—Dr. Worcester's modification of tongs, with blunt conical points to prevent too deep penetration.

shortened condition, and necessitates later a far greater weight than would have been initially needed. The suspension is done in the recognized way, so that active and passive motion (by means of pulleys) of both the hip and knee are at once begun. In humeral fractures, the traction is obtained either by adhesion to the skin or by means of the Henniquen band around the flexed elbow. From five to eight pounds is sufficient, and either method will support this without difficulty. The suspension is by means of a specially constructed addition to the Balkan frame, which permits traction at any angle with the trunk

(Fig. 4). Fractures of the leg are usually drawn into direct alignment without difficulty. Occasionally, however, badly comminuted fractures show a persistent deformity which can be readily overcome by tong traction on the malleoli or os calcis, or by the Henniquen band if low, or skin traction if high. Our success with the Sinclair skate has not beeen satisfying.

What has been stated concerning our satisfaction in this method of treating fractures through the middle third of the

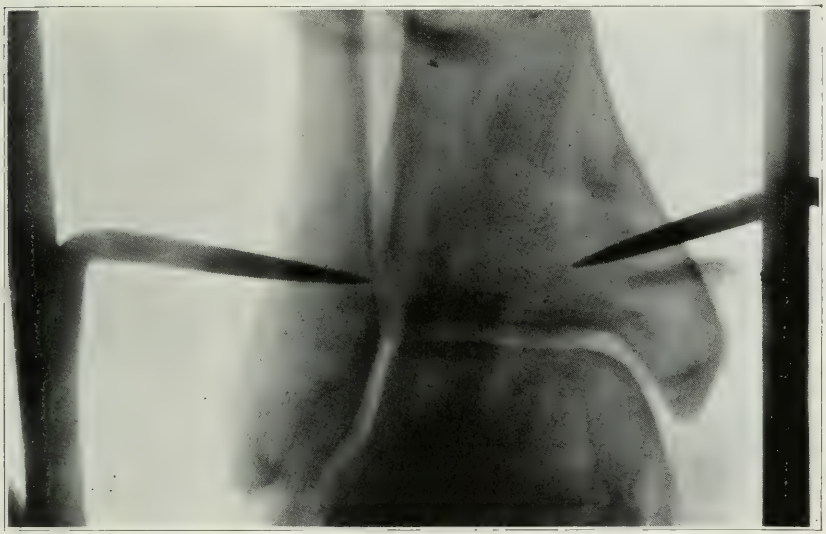

FIG. 2.—Tong points too tapering. They penetrate too deeply.

long bones, cannot be said with the same degree of satisfaction concerning fractures near the large joints. However, our experience here leads us to believe that further study will overcome the difficulties we have encountered, and we are satisfied that in the end such fractures will yield a higher percentage of good results by this method than by any other.

It must be emphasized that perfect anatomic repair of a bone, accompanied by badly crippled joint, is far more disturbing to the patient than poor position of the fragments

with a well-functioning neighboring joint. In many cases a proper reposition of the fragments has in my hands proved impossible. Nature, however, has responded more than satis-

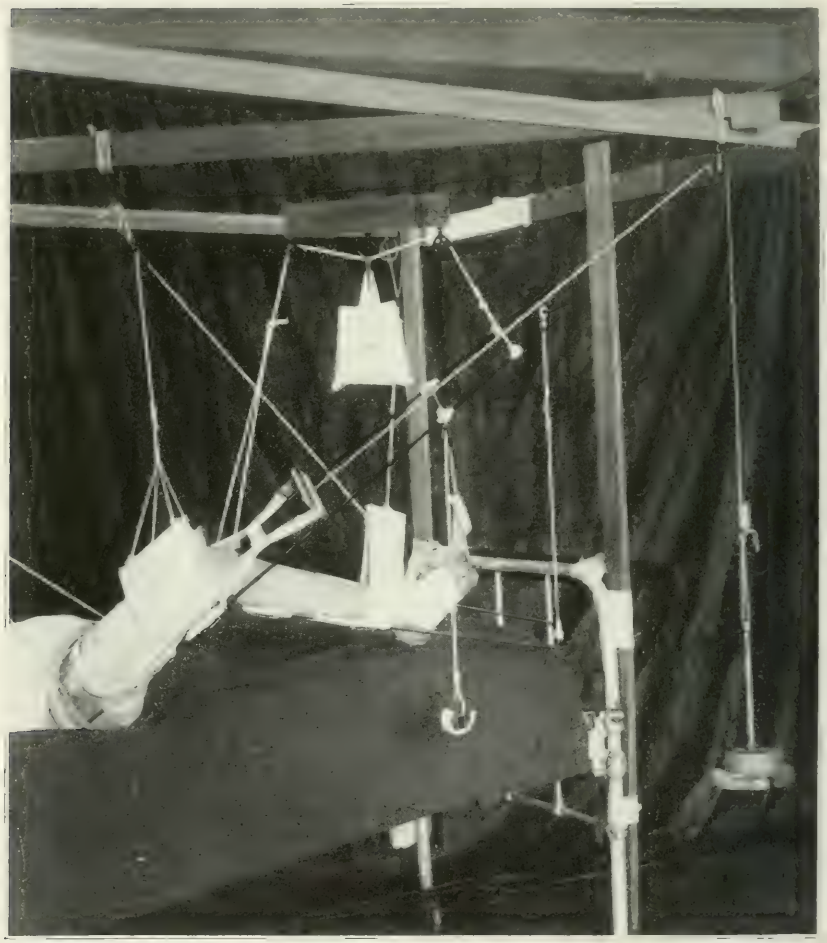

FIG. 3.—Suspension in Balkan frame for a high fracture of the femur with skin traction, illustrating outward rotation, abduction and leg flexion splint attached to the Thomas splint. When only moderate traction is needed, it can be obtained by skin adhesion on the thigh, still allowing knee movements.

factorily in her ability to reëstablish a range of motion in the joint which permits of all essential activities.

Fractures near the hip, the knee, and the shoulder have received the most attention.

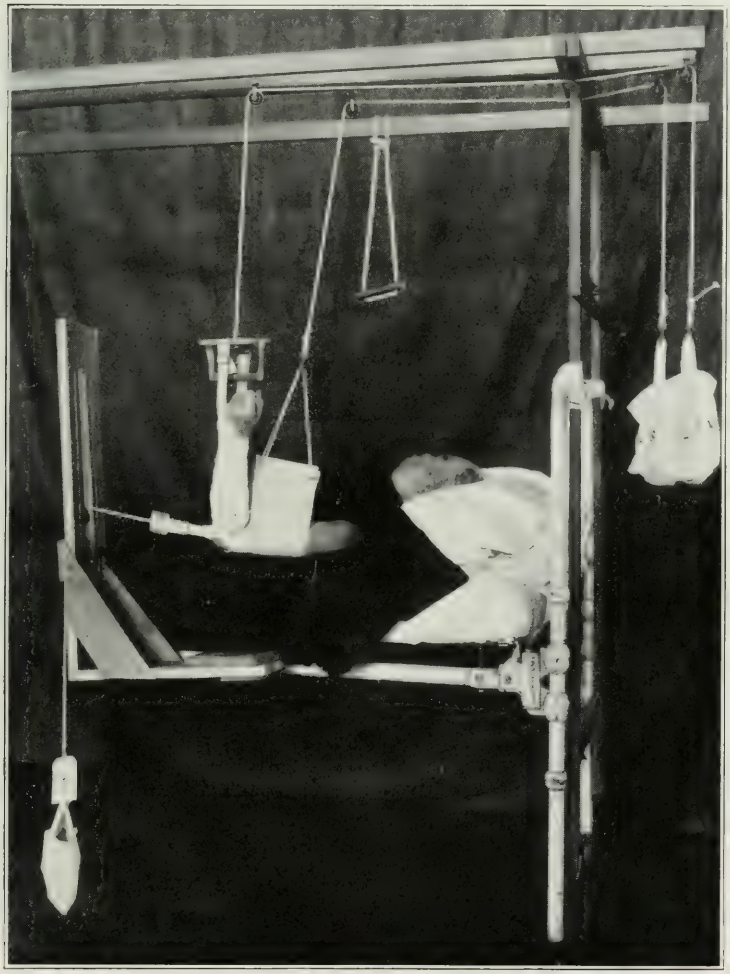

FIG. 4.—Traction of arm in only slight abduction. The amount of abduction is modified as needed by means of the hinge between the mattress board and the horizontal board. The elbow is lowered or raised by the movable pulley attachment within the slot on the vertical support.

When the femur is fractured somewhat below the tuberosities, the upper fragment usually assumes a more or less constant

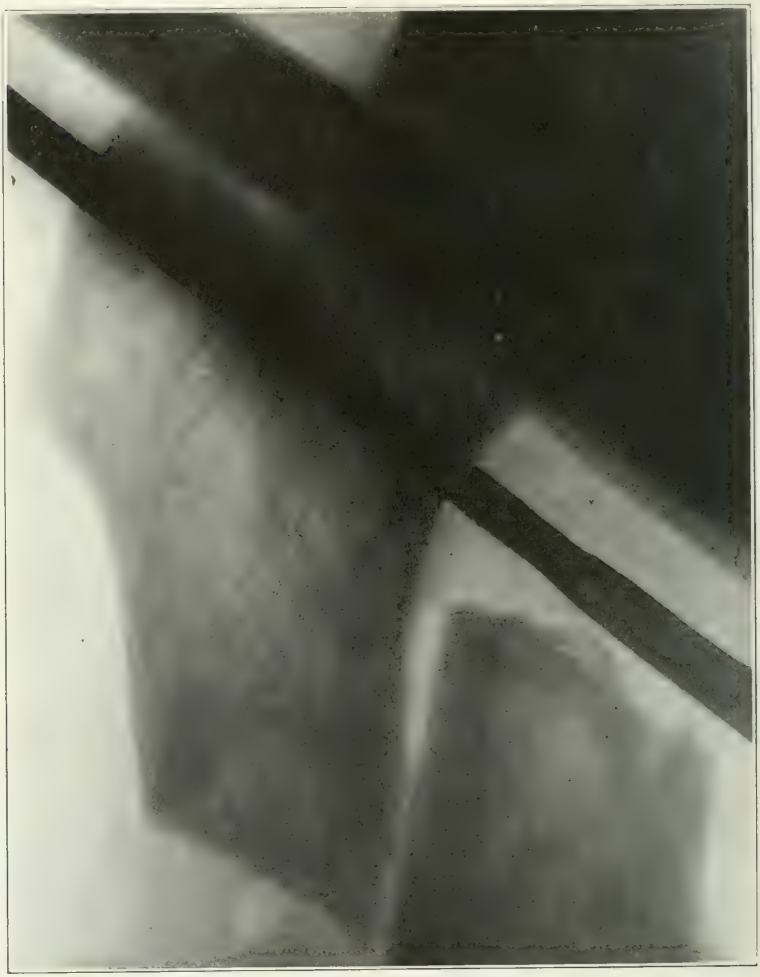

FIG. 5.—Subtrochanteric fracture without either abduction or external rotation of the upper fragment. This fragment is held in slight adduction by the pull on the adductors exerted by the lower fragment being drawn well upward and inward. The fracture was caused by violence applied directly to the external aspect of the femur at the point of fracture, with sufficient force to displace it well inward.

position of external rotation, and abduction, by reason of the stronger group attached at the greater tuberosity, and of flexion because of the direct pull on the lesser trochanter. The extent of these forces varies greatly according as the fracture is near or removed from the line of the tuberosities. If placed some inches distant, so that some fibers of the adduction group remain attached to the upper fragment, this portion of the femur may be drawn into adduction, and neither external nor internal rotation is present. Moreover, in some instances, the fracturing violence may cause unusual positions of the fragments, at the time of injury, the fractured ends having penetrated deeply into the muscle and fascial structures are prevented from taking up the positions resulting from the abnormal muscular pull alone. We have thus come to learn that one may not rely on a knowledge of muscular action alone to predicate the position of the fragments even in such classical fractures as subtrochanteric or those through the surgical neck of the humerus. Dependence must, in the last analysis, be placed on the roentgen ray, and, therefore, the treatment of fractures can be carried out properly only when a bedside apparatus is continuously available.

These points are all well illustrated in the case of a woman, aged sixty years, who, in a street fight, was knocked down and jumped on by another woman (Fig. 5). The violence was applied mainly to the upper right thigh. Admission to the hospital was three days after the injury. There was an obvious fracture about three inches below the trochanters, with overriding of two inches. The upper end of the lower fragment was displaced inward and slightly anteriorly as might be expected. The upper fragment, however, was also adducted, and in normal rotation. A study of the condition, both by palpation and the roentgen ray, showed that the upper fragment was held in this position because of the pull of the torn adductor muscles, which were tightly stretched over the lower fragment. The latter, by the direct violence of the injury, had been forced inward and dragged the upper fragment with it.

An attempt to place such a fracture into proper alignment by abduction of the limb with traction suspension failed. A satisfactory position was obtained only by long traction of twenty-five pounds applied above the condyles, with the limb

FIG. 10.—Same fracture as in Fig. 9, twenty-four hours after applying long traction of 25 pounds. The swelling is somewhat less, the upper fragment is still abducted.

suspended in adduction and some flexion. Even though there was a delay of a week from the time of injury before this was done, the overriding was entirely reduced in forty-eight hours (Fig. 6), whereas five days of skin traction had completely failed.

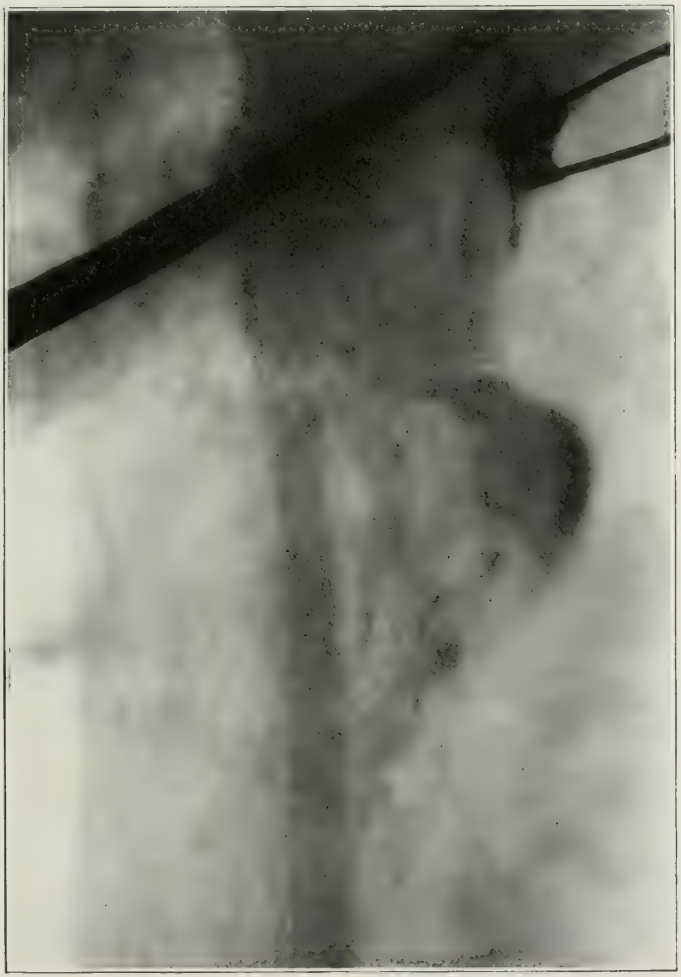

FIG. 7.—Same fracture as in Figs. 5 and 6, showing better alignment which was obtained by placing the limb in adduction, with lateral counter pull by a sling placed around the upper end of the lower fragment.

After correction of the shortening, there was still inward displacement of the upper end of the lower fragment which could be corrected only by direct outward pull by means of sling and pulleys.

FIG. 8.—High spiral fracture of femur together with fracture through the lesser trochanter; upper fragment rotated outward and abducted; satisfactory result obtained by the treatment shown in Fig. 2. In such cases only moderate traction is needed.

The functional result in this patient was most satisfactory. Movement at all joints was practically normal; union was firm, and the anatomic deformity in the bone could be known only by means of the roentgen ray. In consideration of her age, sixty years, her alcoholic habit, and the presence of Paget's disease, she continued to use an ambulatory splint for eight months.

It was this case which first emphasized the necessity of recognizing that dependence cannot be placed on muscle pull alone

Fig. 9.—Pertrochanteric fracture.

in determining the position of the fragments. Several days were wasted in an attempt to place the lower fragment in line with an upper fragment erroneously presupposed to be abducted.

A contrast to this case is shown in Fig. 8. The fracture is higher, the lesser trochanter being attached to the lower fragment, and the line of fracture is spiral. The upper fragment is in marked abduction and outward rotation, the classical deformity. Suspension, tong traction of twenty-five pounds immediately after receipt of injury, reduced to fifteen pounds after

only twenty-four hours, and replaced by skin traction after four weeks, was the treatment used. The limb was placed in 45 degrees abduction, moderate flexion and extreme external rotation. The knee was flexed to 45 degrees on a movable leg attachment to the Thomas splint. There was complete union, with full motion at all joints, at the end of two months. The patient was up in a chair for a month longer and then discharged wearing a Thomas caliper splint. He has failed to report at the follow-up clinic, but there can be no doubt of an excellent result as judged by the condition on discharge.

Our experience with the suspension traction treatment of fractures in immediate relation to the hip-joint, either through the trochanter or of the neck proper, has not been extensive. It is sufficient, however, to convince us that excellent results may be obtained with the former, and we can find no reason to believe that the latter also will not respond favorably.

A pertrochanteric fracture, involving the distal end of the neck, was received by a man, aged thirty-six years, when he fell down-stairs. He was immediately admitted to the hospital with the typical disability and deformity of this condition (Fig. 9). Treatment was by suspension in flexion, outward rotation, and 45 degrees abduction. The knee was placed in 45 degrees flexion on a movable leg attachment to the Thomas splint. Skin traction was used to the amount of fifteen pounds for six weeks, and then reduced to ten pounds. The patient was allowed to be up in a wheel chair with a Thomas splint attachment for suspension and traction (Fig. 10), devised by the resident surgeon, Dr. William A. Rogers, for ten weeks longer. He was then discharged wearing the caliper splint. He reported six months after receipt of the injury, when the following condition was noted: "The patient walks normally. No limp. Has no pain in hip, but states that the knee becomes stiff when he does not use it. After walking a little, it is all right. The caliper brace was removed five and a half months after receipt of the injury." Complete restoration to a normal working condition, therefore, was present in this case in less

than six months. The absence of muscular atrophy and joint disability is a noteworthy concomitant of this form of treatment as compared to their prevalence in complete fixation with plaster cast.

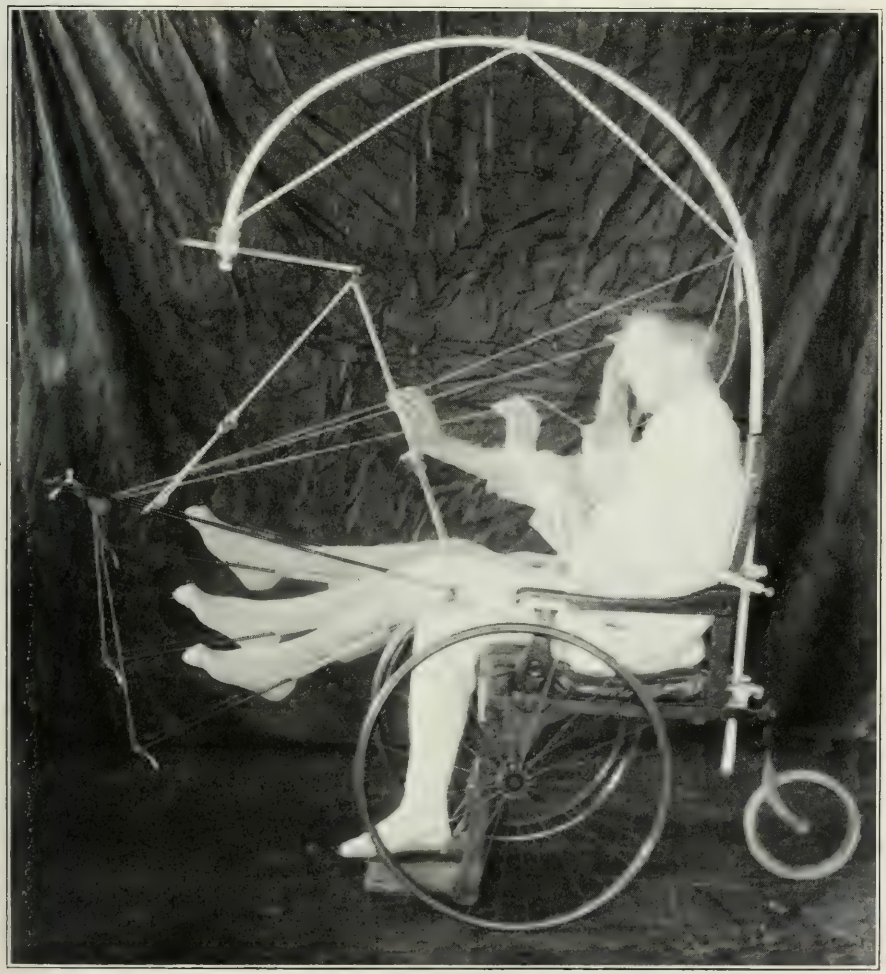

FIG. 10.—Dr. Rogers's attachment to wheel chair for carrying out all the requisites of traction suspension treatment. This should be used only when union is nearly complete and when only moderate traction is needed.

As stated, our experience in similar cases is limited; but there have been no failures, and the consensus of opinion of the staff is that the results are far more satisfactory than with fixation methods, and that the patients are, on the whole, far more comfortable.

FIG. 11.—Fracture through neck of femur, showing result after three months' treatment in traction suspension, with 15 pounds skin traction.

The bone repair of a typical fracture of the neck of the femur is shown in Fig. 11, and the functional outcome at the end of three months is shown in Fig. 12. This fracture was treated

throughout with suspension traction in marked abduction and external rotation. Skin adhesion was used, and movements in the hip and knee were carried out from the first. The result, after six months, was an entirely normal hip so far as weight

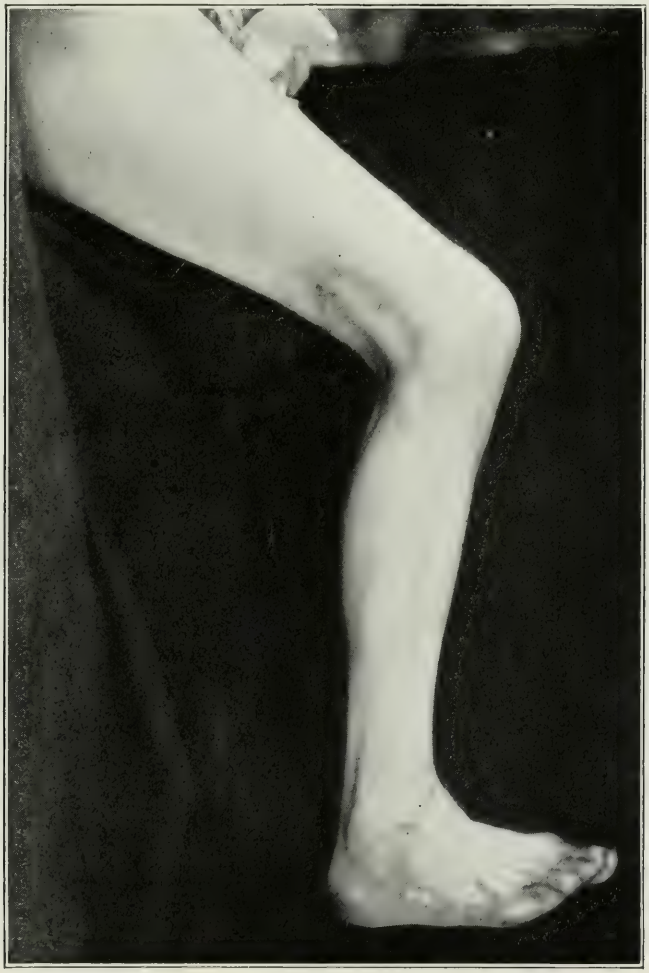

FIG. 12.—Fracture through neck of femur, showing functional result after three months' treatment in traction suspension, with 15 pounds skin traction.

bearing and range of motion were concerned. The roentgenogram showed a moderate coxa vara.

FIG. 13.—Fracture of surgical neck of humerus, with the greater tuberosity completely avulsed. The upper fragment is turned so that the fracture line looks almost inward. This is hardly possible if the tuberosity remains attached to the proximal fragment. This patient was operated on with fair success.

Fractures in and about the shoulder-joint show many similarities to those occurring in the upper end of the femur. The

FIG. 14.—Extreme abduction and external rotation needed to procure proper alignment when a surgical neck fracture allows this exaggerated position of the proximal fragment by pull on the greater tuberosity.

essential difference, however, in obtaining a satisfactory functional result is that the range of movement in the shoulder is all

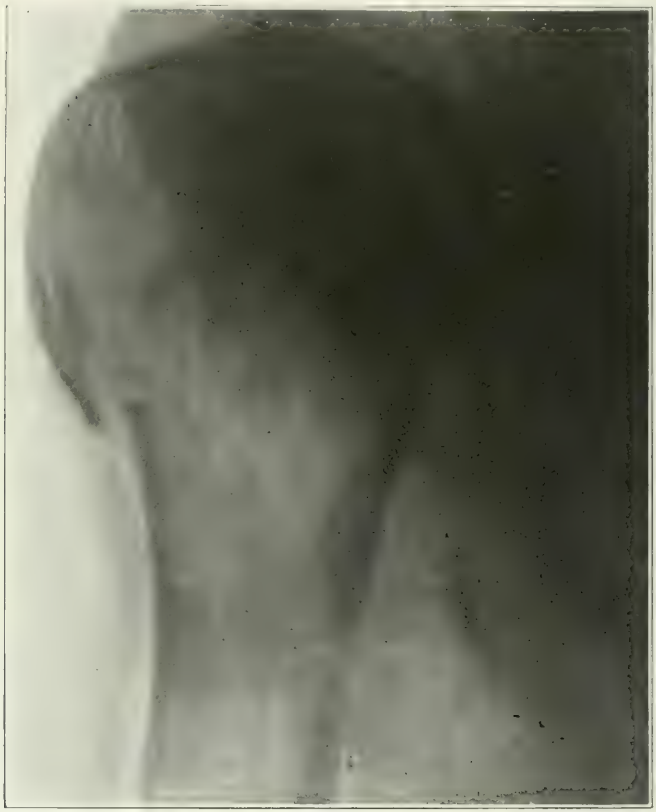

FIG. 15.—Upper fragment in surgical neck fracture is not abducted or rotated, because, as we believe, the muscular attachments are torn away without actual separation of the tuberosity. This fracture was at first treated in slight abduction and later full abduction and external rotation (Figs. 4 and 14 are examples) with excellent result and full restoration of function.

important, while weight bearing without shortening is the prime consideration at the hip. For this reason, treatment of high fractures of the humerus must aim to procure union with the

fragments so placed that joint obstruction will not take place and that muscular action may be efficient.

The usual deformity which results from a fracture through the surgical neck of the humerus is abduction and external rotation of the upper fragment. There are many exceptions, however, due to various causes. The most common is a coincident tearing off of the greater tuberosity. This leaves the upper fragment uninfluenced by the muscles attached to this prominence, and it may assume any position, even to an amount of rotation inward so that the fractured surface faces the glenoid fossa. Such a complete reversal results in leaving the upper fragment beyond control, and so far as we have determined, nothing can be accomplished by traction suspension in changing its position (Fig. 13). Open operation seems a necessity. On occasion, however, the inward displacement is not so complete, and then by placing the humeral shaft in the proper amount of abduction, the fragments are well aligned. After from ten days to two weeks, there is sufficient union to cause the upper fragment to follow the arm as the abduction is increased from day to day. Finally, there is hyperabduction and external rotation which then bring the upper fragment in contact with the abducted and externally rotated greater tuberosity. Union then results. On recovery the arm falls by gravity to adduction and internal rotation, by which the abduction and external rotation groups of muscles are drawn out to their normal length, and hence can again function. It is important that some movement should be made in the arm constantly. The traction suspension treatment ideally furnishes an opportunity for this. Ten pounds is usually sufficient, applied by means of skin traction. The needed modifications in position are obtained by means of the support (Fig. 4) and direct traction (Fig. 14).

One must further recognize that inward rotation of the upper fragment may take place when the muscles or the periosteum are torn from the greater tuberosity without actual separation of the tuberosity itself. This injury need not show in the roentgenogram, though in some cases the periosteal stripping

can be seen (Fig. 15). The importance of this is found in the fact that unless the inward rotation is recognized, the arm may

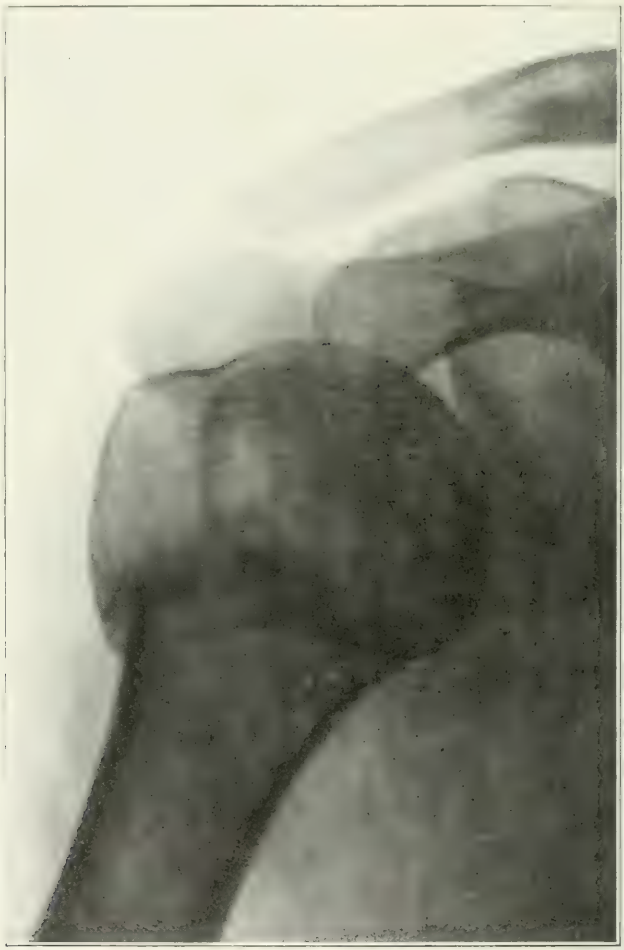

FIG. 16.—Variations in position in fracture of surgical neck with tearing away of muscles from greater tuberosity.

be easily placed in too great abduction. We have fallen into this error on one or two occasions.

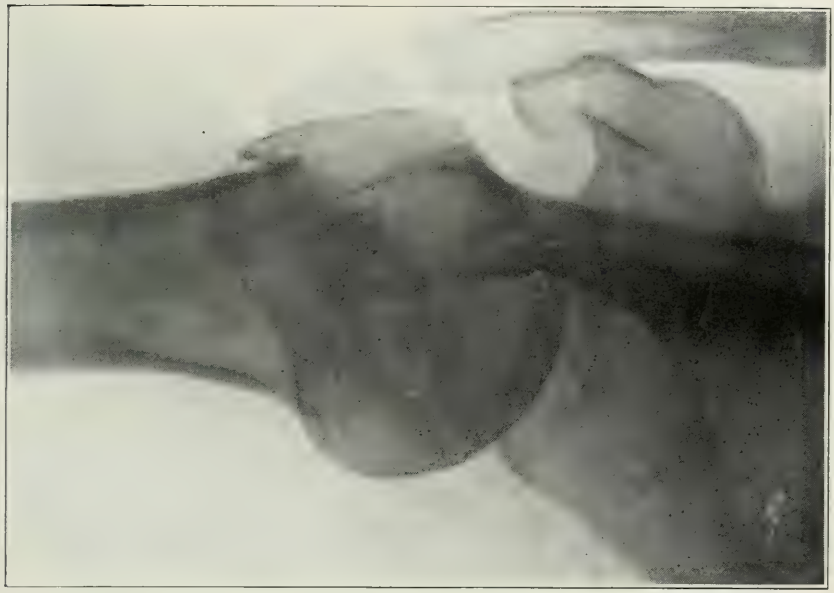

FIG. 17.—Variations in position in fracture of surgical neck with tearing away of muscles from greater tuberosity.

FIG. 18.—High spiral and comminuted fracture of the humerus, in abduction after partial union is established. The proximal fragment was drawn inward by pectoral major fibers.

Here again, when the fragments are sufficiently adhered, it is necessary to increase the abduction from day to day, so that

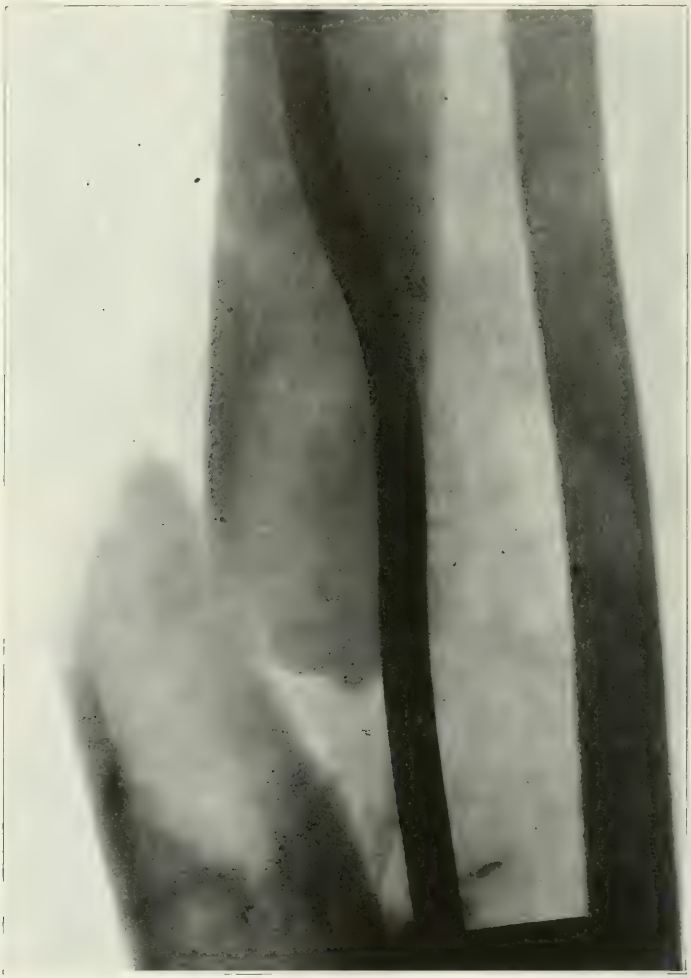

FIG. 19.—Fracture near knee-joint, with the lower fragment forward. Flexion of knee exaggerated this deformity while extension corrected it. Fig. 20 represents the same amount of traction (25 pounds) by long attachment, in extension.

the greater tuberosity may be brought into contact with its separated and misplaced muscular insertions.

Finally, the fracture of the surgical neck may run obliquely down the shaft so that portions of the pectoral major are attached

FIG. 20.—Fracture near knee-joint, representing the same amount of traction (25 pounds) by tong attachment as Fig. 19, which is in flexion, while Fig. 20 is in extension.

to the upper fragment. This condition, with entanglement of the fragment ends in this muscle and the deltoid or arm muscles, may produce bizarre positions.

FIG. 21.—Late result: some deformity, but fair movement in the knee-joint.

The important consideration, therefore, is to obtain accurate information as to the position of the upper fragment, by means

of the roentgen ray—no other examination will give this—and place the distal fragment by traction suspension in proper

FIG. 22.—Late result: some deformity, but fair movement in the knee-joint.

alignment with it. Ultimately, however, the extremity must be brought into complete abduction—sometimes hyperabduction—and external rotation. During healing, a certain amount

of active motion must be carried out, this being facilitated by proper counterpoising, so that the entire weight of the limb does not fall on the muscular pull.

The service has afforded many examples of these fractures, all

FIG. 23.—Fracture treated by suspension and traction by tongs in the os calcis, with excellent result.

of which have been treated along the lines here described. The functional results have been, without exception, excellent.

This form of treatment, of course, requires confinement to bed for a period of about four weeks. This is not too great a

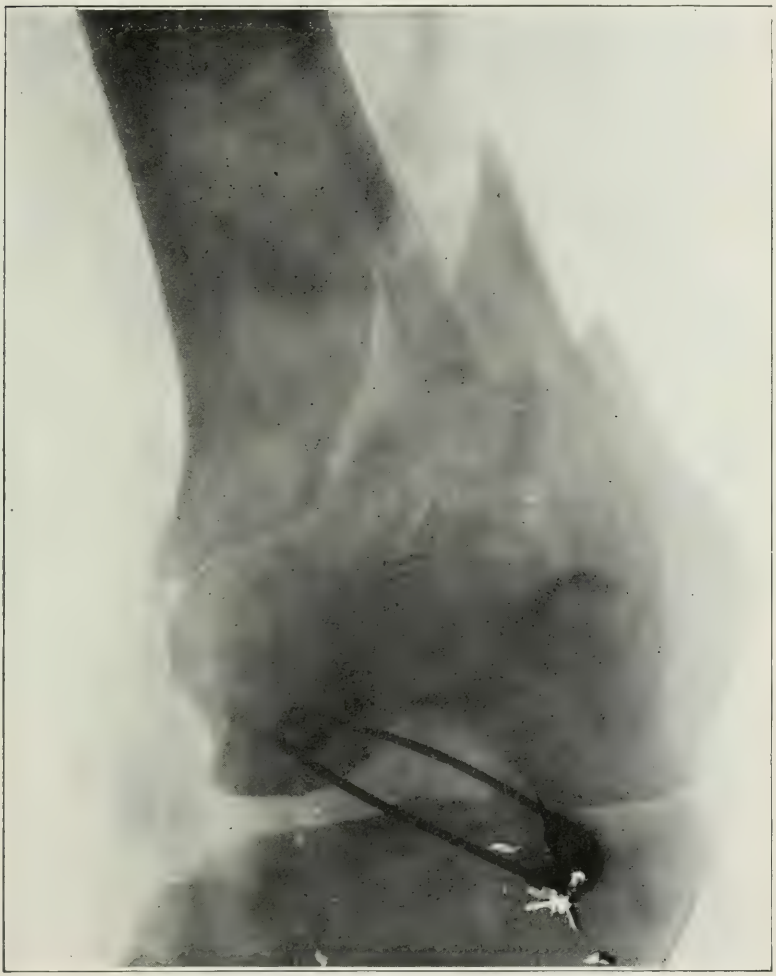

FIG. 24.—Comminuted fracture about femoral condyles, treated by suspension and traction on Steinmann pin through tibial head, for five weeks, with leg extended. Active motion on movable leg attachment then instituted.

price to pay for the obtaining of a thoroughly useful shoulder. Examples of these fractures are given in Figs. 11 to 18, and the

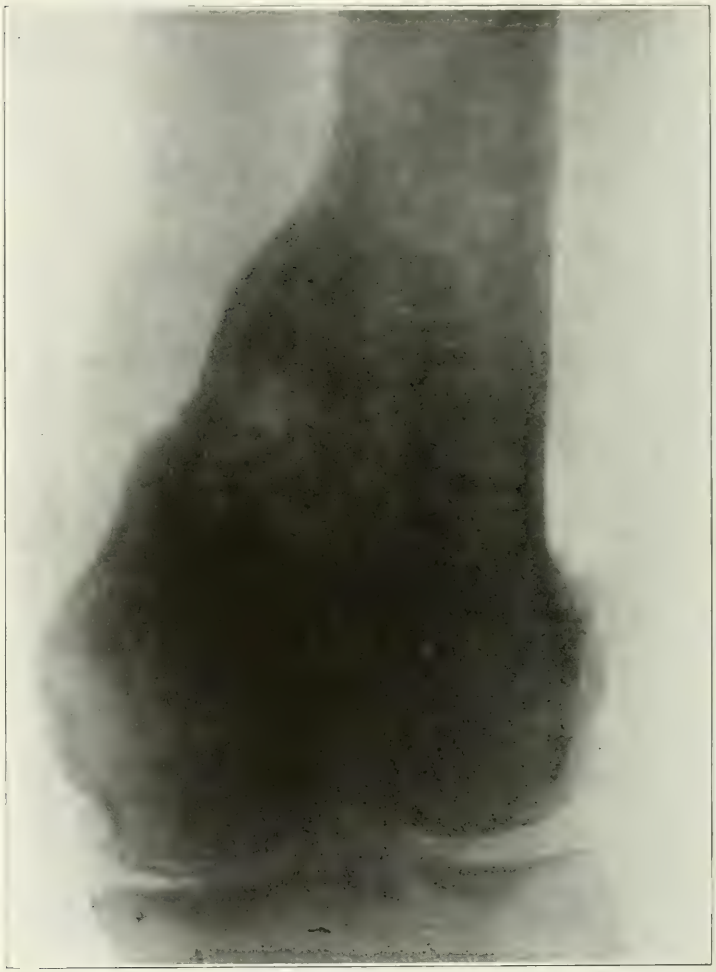

FIG. 25.—The result.

methods of obtaining the needed positions are illustrated in Figs. 4 and 14.

Fractures of the femur near and involving the condyles present peculiar difficulties. The typical supracondylar fracture, with the lower fragment drawn directly backward, can easily be drawn into proper alignment by placing the tongs sufficiently far back and high enough to draw this fragment forward. This is accomplished by so arranging the supporting pulley that the line of traction is 45 degrees or more upward. The fulcrum then comes at the line of the condylar surface against the patella and

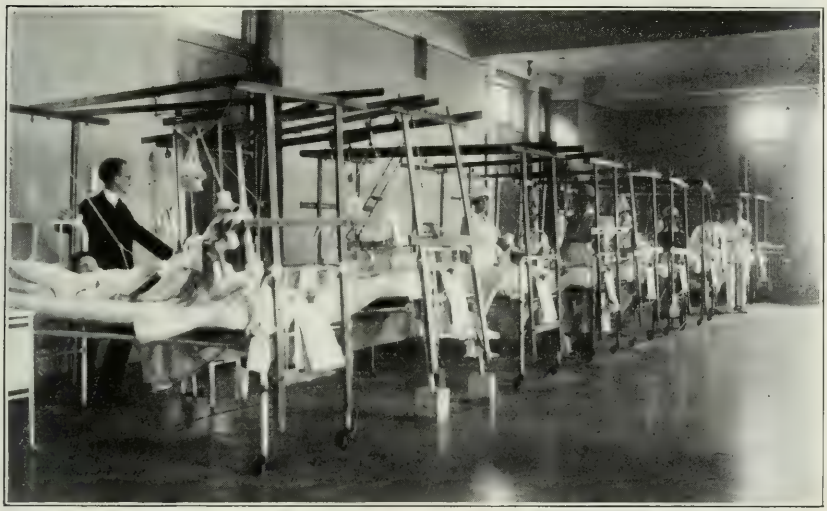

FIG. 26.—The fracture ward, showing the number of cases on a general surgical service of ninety beds.

its ligament. This is made more efficient by flexion of the leg, and in the first days movements in this joint may be only minimal. After some days, however, the alignment can be maintained even when the leg is moved through a considerable arc. When, however, as illustrated in Fig. 19, the lower fragment, by the fracturing force, is driven forward, while the upper fragment lags behind, we have been able to get proper alignment only by keeping the leg in extension. To do this sacrifices one of the great advantages of the method of treatment, namely, active

movement. One must, therefore, determine in each case whether the wise course is to obtain good alignment without active motion in the knee, or accept some deformity with a good functioning knee-joint. In the case above cited, a middle course was followed with a fairly satisfactory result (Figs. 21 and 22), though the fracture was compound, with almost complete rupture of the quadriceps at the site of fracture.

FIG. 27.—The fracture truck assembled by Dr. Bulkley. This carries everything needed in applying suspension traction and is a most essential unit in the equipment.

The badly comminuted and displaced fractures involving the femoral condyles and knee-joint present unusual difficulties. As a rule only moderate traction is needed; but the problem of

securing this, and at the same time of permitting knee movement, must be solved. Fig. 24 shows such a fracture in a woman, aged sixty years, received by a fall of eight feet and landing on the foot with leg extended. The treatment was by means of traction on the Steinmann pin through the tibial head, and suspension at first with the leg extended. After five weeks the movable leg attachment was utilized, traction removed and active movement at the knee instituted. The result is shown in Fig. 25, when the function of the knee was excellent and the patient could walk without pain or limping. Flexion beyond a right angle was present.

No other similar case has come under treatment, but the outcome here indicates that the suspension traction treatment can be used to advantage.

The suspension traction treatment has also been found efficient in badly comminuted or long spiral fractures of the tibia and fibula near the ankle-joint. The tongs may be placed either in the malleoli or the os calcis. Fig. 23 shows one case of this sort treated with an excellent result.

Our experience has led to the conviction that this method of treatment is deserving of careful study on the part of those treating fractures. In a hospital where fractures are treated in any number, it should constitute the standard treatment. Its possibilities, I believe, are greater than the results here shown. It is not, however, a method easy of use, and only by rather long intensive study may one perfect himself in the technic.

The study on the Bellevue service has been carried on by the entire staff. I desire particularly to express my appreciation to my former associate surgeons, Dr. Kenneth Bulkley and Dr. James Worcester, who successively have had charge of the fracture work, and to the resident surgeon, Dr. Russell Patterson, who has taken most of the roentgenograms and given valuable aid in the preparation of this communication.

CLASSIFICATION AND MECHANISM OF FRACTURES OF THE LEG BONES INVOLVING THE ANKLE

BASED ON A STUDY OF THREE HUNDRED CASES FROM THE EPISCOPAL HOSPITAL

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AND

RALPH S. BROMER, M.D.

PHILADELPHIA

(By Invitation.)

Anatomical and Surgical Study

By Dr. Ashhurst

Pott, Dupuytren, Cooper, Maisonneuve, Tillaux, Hönigschmied, Stimson, Destot, Chaput, Quénu—and shall I not add Scudder, Cotton, Roberts and Speed? What can any one say more, at this late day? And yet the fact remains that there is no entirely satisfactory classification of ankle fractures in existence, and that many points of the mechanism of their production still are in dispute. I say there is no entirely satisfactory classification of these fractures in existence, because that which is the best, being the most scientific and complete, namely that of Quénu, is less a classification than a catalogue; and because his strict adherence to an anatomopathologic classification and his stern rejection of the historical pathogenetic classification compel him to place side by side lesions in no sense related, except that the fracture lines happen to be similar, and to separate widely other lesions which, though presenting very dissimilar

lines of fracture, nevertheless represent only different degrees or stages, or merely variants, of one and the same lesion.

It is perhaps unnecessary to argue the desirability of classification, for without classification the relation of one lesion to another can be neither remembered nor understood in any department of knowledge; and comprehension is a prerequisite for intelligent memory and for rational diagnosis and treatment.

HISTORICAL

Si chirurgien experimenté que vous soyez, ne sautez pas ces premières pages. Elles sont indispensables, mais fatigantes à lire; mieux vaut remettre à un autre jour cette besogne ardue que de l'entreprendre mal disposé.—FARABEUF.

Pott[1] (1769) described a fracture which does not exist, and Dupuytren[2] (1819) commended him for his acute observation and fidelity to nature. Cooper[3] (1822), more sensible than either, merely recounted what he had seen, avoided speculation about what he had not seen, and was silent on subjects about which he had no knowledge. Maisonneuve[4] (1840) and Tillaux[5] (1872) studied the mechanism by experiments on the cadaver, and though they were both correct, they came to different conclusions; and Tillaux thought his work had entirely invalidated that of Maisonneuve. Hönigschmied's[6] (1877) experiments on cadavers (125 in all) may be said to have carried this means of investigation to its limit; and though subsequent students, including myself, have repeated such experiments, it is evident that this method of investigation has many limitations, as it is impossible to reproduce in the cadaver the falls which living patients suffer and the muscular tension to which their limbs are constantly subjected.

[1] Some Few General Remarks on Fractures and Dislocations, London, 1769, p. 57.
[2] Ann. méd.-chir. d. hôp. et hosp. civ. de Paris, 1819, p. 1.
[3] Treatise on Dislocations and Fractures of the Joints, London, 1822.
[4] Arch. gén. de méd., 1840, i, 165, 433.
[5] Cited by Gosselin: Bull. de l'Acad. de méd., Paris, Series 2, 1872, i, 817.
[6] Deutsch. Ztschr. f. Chir., 1877, viii, 239.

Stimson's[7] study, in 1892, was the last one of importance before the advent of the roentgen ray; and it was Destot's[8] (1911) who first of all published extensive observations of ankle fractures illustrated by, and based on, roentgenographic studies. It is strange that no English writer since Cooper has made a special study of fractures at the ankle, and that with the exception of Stimson, already mentioned, no American surgeon has made a particular study of the subject, though Scudder, Cotton, Roberts and Kelly, and Speed have discussed it at greater or less length in their text-books on fractures. It is evident, however, to any attentive student of the subject that most of the recent writers, if not, indeed, all, demonstrate by their statements that they have not themselves read, or at any rate have not read with understanding, the works of their predecessors—especially of Dupuytren, Cooper, Maisonneuve and Tillaux. Most surgeons think they know what they mean by a "Pott's fracture," but, as I said before, there is little use in knowing much, or anything, about a type of fracture which does not exist.

What is Pott's fracture? Let Mr. Percivall Pott tell in his own words[9] (1769).

"The limb most commonly preserves its figure and length . . . if the fibula only be broken, in all that part of it which is superior to letter A in the annexed figure [Fig. 1], or in any part of it between its upper extremity, and within two or three inches of its lower one. . . . I have already said . . . that the support of the body and the due and proper use . . . of the joint of the ancle depend almost entirely on the perpendicular bearing of the tibia upon the astragalus, and on its firm connection with the fibula. If either of these be perverted or prevented, so that the former bone is forced from its just and perpendicular position on the astragalus, or if it be separated by violence from its connection with the latter, the joint of the ancle will suffer a partial dislocation internally; which

[7] New York M. J., 1892, lv, 701.

[8] Traumatismes du pied et rayons-x, Paris, 1911.

[9] It should be borne in mind that Pott is discussing the importance of treating fractures of the leg bones in the flexed position (the limb resting on its outer surface with the knee bent), and hence that he speaks of fractures of the fibula only incidentally.

partial dislocation cannot happen without not only a considerable extension or perhaps laceration of the bursal ligament of the joint, which is lax and weak, but a laceration of those strong tendinous ligaments which connect the lower end of the tibia with the astragalus and os calcis, and which constitute in great measure the ligamentous strength of the joint of the ancle.

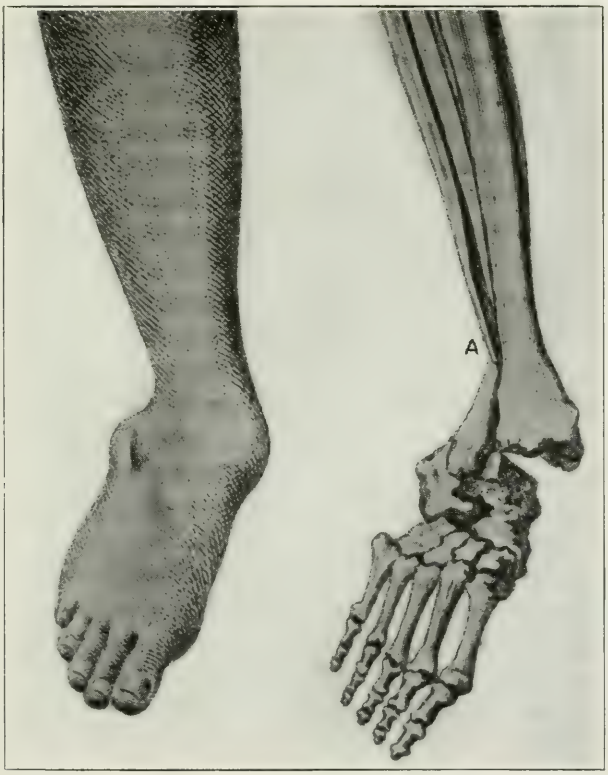


FIG. 1—Illustration from Pott's work, *Some Few General Remarks on Fractures and Dislocations*, London, 1769, facing p. 69.

"This is the case when, by leaping or jumping, the fibula breaks in the weak part already mentioned, that is, within two or three inches of its lower extremity. When this happens, the inferior fractured end of the fibula falls inward, toward the tibia, that extremity of the bone which forms the outer ancle is turned somewhat outward and upward, and the tibia, having lost its proper support and not being

of itself capable of steadily preserving its true perpendicular bearing, is forced off from the astragalus inward [Evidently, he means by the weight of the body, after primary fracture of the fibula produced by the first impact of the foot with the ground], by which means the weak bursal, or common ligament of the joint is violently stretched, if not torn, and the strong ones, which fasten the tibia to the astragalus and os calcis, are always lacerated, thus producing at the same time a perfect fracture and a partial dislocation, to which is sometimes added a wound in the integuments, made by the bone at the inner ancle. By this means, and indeed as a necessary consequence, all the tendons which pass behind or under, or are attached to the extremities of the tibia and fibula, or os calcis, have their natural direction and disposition so altered, that instead of performing their appointed actions, they all contribute to the distortion of the foot, and that by turning it outward and upward."

[Note that there is not a word of posterior displacement, and that this is not shown in the plate, here reproduced as Fig. 1.]

This description, and the accompanying illustration, call for several remarks. It is to be noted, first, that Pott's fracture, as described and pictured by himself, is a primary, nearly transverse, fracture of the fibula, attended by a subsequently produced "partial dislocaton" of the ankle-joint internally. Second, the fracture of the fibula occurs at, or below, the point marked *A* in the illlustration ("within two or three inches of its lower extremity"); and the upper end of the lower fragment is described as falling in against the tibia, while the external malleolus is turned outward. Third, there then is supposed to occur rupture of the ligaments below the internal malleolus, with a partial dislocation of the tibia inward, off the trochlea of the astragalus, as the latter bone turns outward, around a more or less anteroposterior axis. Now, such a fracture of the fibula does not occur as a type, as may be readily verified by the examination of any series of roentgenograms or post-mortem specimens; and if it did occur, it would be impossible for the upper end of the lower fragment to fall in against the tibia, for the reason that the fibula is already closely applied to the tibia at the point described. There is barely room to insert the blade

of a scalpel between the bones at a point three inches (7.5 cm.), or less, above the tip of the fibula.

It was the particular merit of Dupuytren to systematize the teaching of various pioneers, such as Pott (1769), Bazille[10] (1771), Bromfeild[11] (1773) and Pouteau[12] (1783), who had recognized, as fractures, lesions which had been regarded by former generations as dislocations; and it is to Dupuytren's memoir on "The Fracture of the Lower End of the Fibula"[13] that surgery is principally indebted for its understanding of all these ankle fractures. It is true that Gosselin (1872) reproached Dupuytren with having propounded his theories of mechanism merely by means of his reasoning, and not on a basis of clinical observations or cadaveric experiments (though Dupuytren made a number of the latter, and presented records of 207 patients with ankle fractures). But there is much justification for Dupuytren's[14] statement that the mechanism detailed by patients cannot be relied on, since it is well known that in dislocations of the shoulder, for instance, they all will say the injury resulted from a fall on the point of the shoulder (because that is where they feel the pain), whereas the state of their elbow or hand proves quite the contrary. Similarly, I have known a patient with an ankle fracture badly united in abduction (fibular flexion) to assert that it was produced by adduction (tibial flexion), because her heel was prominent beneath the internal malleolus just after the accident; not realizing that the heel had been brought into

[10] Mém. sur les sujets proposés pour le prix de l'Acad. Roy. de Chir., Paris, 1778, iv, 563.

[11] Chirurgical Observations and Cases, London, 1773, ii, 78.

[12] Oeuvres posthumes, Paris, 1783, ii, 267.

[13] This memoir was said by Nélaton (Eléments de path. chir., Paris, 1844, i, 810), to have been read by Dupuytren in 1813 before the Académie des sciences. However, I have searched the "Procès-verbaux des séances de l'Académie des sciences" (Paris, 1812-1815, v,) and find no reference to such an event; and Quénu (Rev. de Chir., 1912, xlv, 5 (footnote 2, states that he has had the original records of the Academy searched page by page, without finding any trace of Dupuytren's Memoir; and says that "until further information" he will consider as "mémoire princeps" the appearance of the essay in the "Annuaire medico-chirurgicale des hôpitaux et hospices civiles de Paris," 1819, p. 1. The essay is most easily accessible where first reprinted, in the second edition of Dupuytren's "Leçons orales" (Paris, 1839, i, 275.)

[14] Leçons orales de clinique chir., ed. 2, Paris, 1839, i, 328.

that position by outward displacement of the point of the foot, the astragalus turning around the long axis of the leg.

Now it is to be noted that Dupuytren, who commended Pott for his accurate delineation of the typical fracture of the lower end of the fibula, was evidently of the belief that the typical fracture which he himself was describing was the same as that of Pott; and that this is still the opinion of the French is manifest from a footnote of Quénu,[15] in which he says that the French mean by Dupuytren's fracture precisely what the English mean by Pott's. So that it is somewhat confusing to have J. Hutchinson, Jr.,[16] and some recent writers name and illustrate as Dupuytren's fracture a lesion which Dupuytren[17] encountered only once in more than 200 cases, and which consisted in a fracture of the fibula, rupture of the tibiofibular ligaments, and displacement upward of the astragalus along the fibular side of the tibia. As it is a fact that the fracture at the ankle most often seen, and therefore the most typical fracture, is that first described accurately by Maisonneuve (1840),[18] it is safe to assume that both Pott's and Dupuytren's descriptions applied to this fracture and not to the rather hypothetic type they thought they were describing.

However, there is a typical, though rare, fracture at the ankle which corresponds closely enough to the original illustration of Pott to make it worthy of being called by his name; it is a typical "flexion fracture" (Biegungsbruch) of the fibula, usually 8 cm. (three and one-fourth inches), or *higher*, above the tip of the external malleolus, accompanied by fracture of the internal malleolus and almost invariably by rupture of the inferior tibiofibular ligaments, permitting a diastasis between tibia and fibula. It is such a fracture as is illustrated in Fig. 26, and

[15] Rev. de chir., 1912, xlvi, 367 (footnote 2).
[16] Tr. Path. Soc., London, 1887-1888, xxxix, 238.
[17] Footnote 14, p. 649.
[18] Though, unfortunately, it is not known by his name, which is attached to a fracture produced experimentally by him on the cadaver, but which he never saw clinically. The most frequent and most typical fracture the French now call the "low Dupuytren," to distinguish it from the true Dupuytren or Pott fracture, which they name "Dupuytren type."

may well be called Pott's fracture, even though Pott in his own description placed the seat of the fibular fracture too low, and ignored the fracture of the internal malleolus and the tibiofibular diastasis.

Cooper[3] (1822) did not get away from the idea of the paramount importance of the dislocation of the tibia in these lesions (nor, it may be remarked, did Malgaigne, Hamilton, Treves, or Stimson, many years later); and he described the fractures as mere incidents. But any modern surgeon will find it difficult to bring forth as extensive a list of severe ankle lesions as that recorded by Cooper, though he can easily excel Cooper in the number of lesions which (owing to the absence of deformity) can be certainly recognized only with the aid of the roentgen ray.

Cooper describes succinctly, but accurately, the following main groups of lesions; and scarcely one additional type has been discovered since, even with the aid of roentgenography:

1. *Simple Dislocation of the Tibia Inward.* In this case there is a fracture of the fibula, two inches above its tip, carrying with it an attached fragment of the tibia;[19] the lower end of the upper fragment of the fibula rests on top of the astragalus,[20] and the tibia with the internal malleolus intact descends on the median surface of the astragalus. (This is illustrated in Fig. 1 of Plate 16 of Cooper's monograph, first edition, 1822, and is reproduced here as Fig. 2. The figure was copied by Vidal de Cassis[21] in his second edition [1846] as Fig. 53 and the lesion was for many years known in France by his name; until Quénu recently called attention to the fact, quite clearly stated by Vidal, that the illustration was copied from Cooper.) This, says Cooper, is the most frequent of the dislocations of the ankle. It corresponds probably to the form now known to the French as the "low Dupuytren" fracture, though in the latter type,

[19] This is the "intermediate fragment" known to the French as the "third fragment of Tillaux," who produced it frequently in his cadaveric experiments.

[20] Richet (Unión méd., 1875, xx, 142) described such a case in which the astragalus was penetrated by the upper fragment of the fibula. Roentgenograms in anteroposterior view often show such an appearance, but the lateral views I have seen have always demonstrated that the fibula was behind, or very rarely in front of, the astragalus. Richet's lesion was demonstrated at necropsy.

[21] Traité de pathologie externe, ed. 2, Paris, 1846, ii, 394.

which is very frequent, there is very seldom any intermediate fragment detached from the tibia.

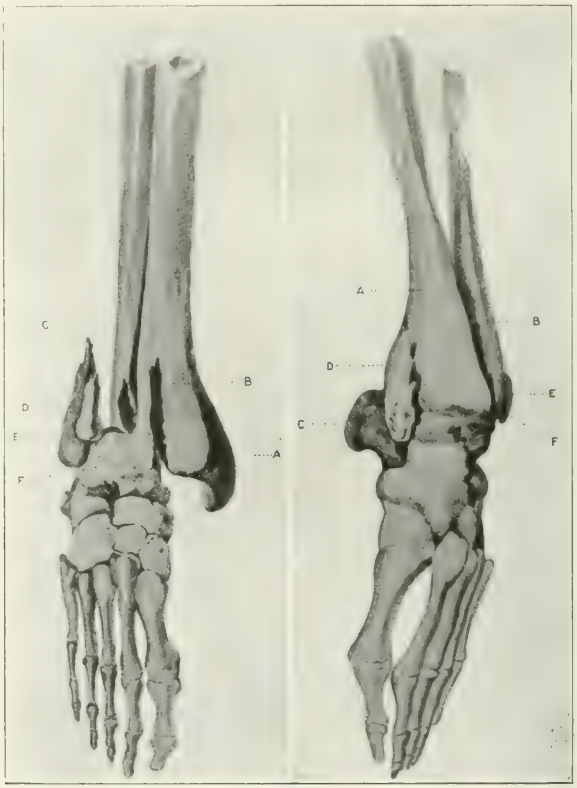


FIG. 2.—Illustrations from Cooper's *Treatise on Dislocations and Fractures of the Joints*, London, 1822, Plate XVI, Figs. 1 and 2. "Fig. 1 shows the dislocation of the tibia inward at the ankle-joint: *A*, the malleolus internus of the tibia thrown on the inner side of the astragalus; *B*, a portion of the tibia split off; *C*, fibula broken; *D*, the broken portion of the tibia adhering by ligament to the fibula; *E*, the malleolus externus of the fibula, with the broken portion of the tibia adhering to it, and *F*, astragalus thrown outward. Fig. 2 shows the dislocation of the tibia outward, at the ankle-joint: *A*, the tibia; *B*, the fibula; *C*, the os calcis; *D*, fracture of the tibia at the malleolus internus, which has become reunited; *E*, extremity of the fibula broken, and *F*, tibia thrown on the outer side of the articulatory surface of the astragalus, to which it is anchylosed."

2. *Simple Dislocation of the Tibia Forward.* In this case there is a fracture of the fibula, three inches above its tip; the internal lateral

ligament is partly lacerated; the tibia and upper fragment of the fibula advance forward, and the tibia rests on the upper surface of the scaphoid and internal cuneiform. In *partial dislocation of the tibia forward*, the articular surface of the tibia is divided in two; the

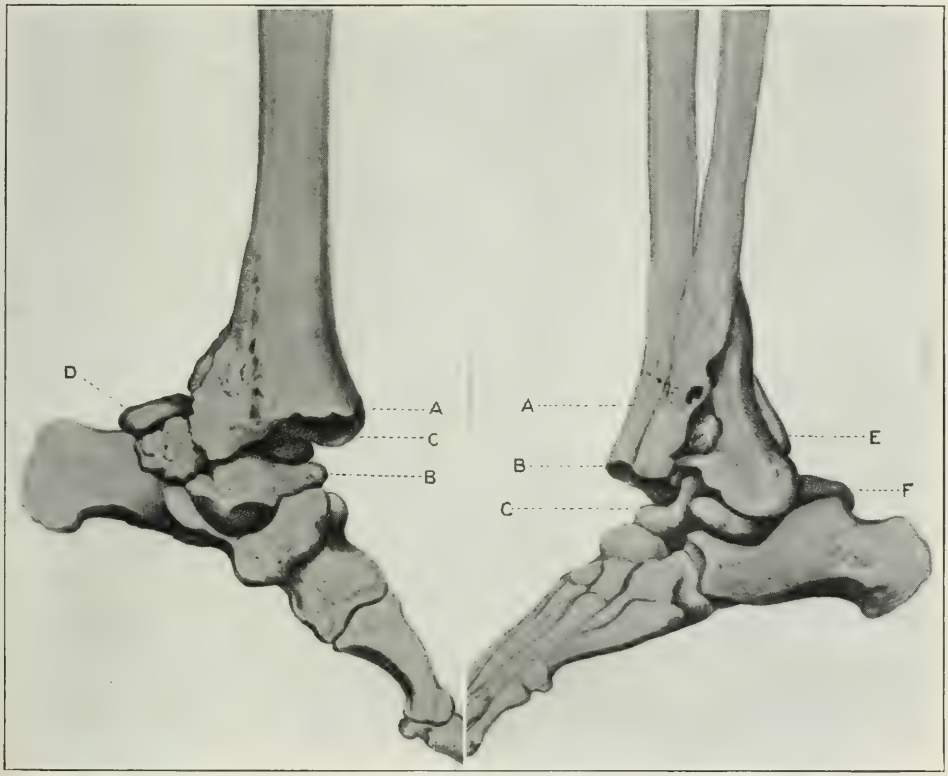


FIG. 3—Cooper's Plate XVII, Figs. 1 and 2, "Partial dislocation of the tibia forwards, at the ankle-joint: Fig. 1: *A*, the tibia thrown forward over the os naviculare; *B*, the astragalus; *C*, new articulatory surface of the tibia, and *D*, the portion of the astragalus behind the tibia. Fig. 2: opposite view of Fig. 1: *A*, the tibia thrown forwards; *B*, new articulatory surface of the tibia; *C*, astragalus; *D*, fibula broken and reunited; *E*, malleolus externus of the fibula, and *F*, astragalus behind the tibia."

anterior part rests on the scaphoid and the posterior on the astragalus. The fracture of the fibula (as shown in Plate 17, Figs. 1 and 2, of Cooper's monograph, reproduced here as Fig. 3) runs obliquely up and back, through the inferior tibiofibular joint. Thus there is

here accurately described the posterior marginal fragment of the tibia, which many recent writers think they were themselves the first to discover after the introduction of roentgenography (Destot, Chaput, Cotton, Sear, etc.).

3. *Simple Dislocation of the Tibia Outward.* This, says Cooper, is the most dangerous of the three, for it is produced by greater violence, etc. The internal malleolus is obliquely fractured and separated from the shaft of the bone; the fractured portion sometimes consists only of the malleolus; at other times, the fracture passes obliquely through the articular surface of the tibia, which is thrown forward and outward on the astragalus before the external malleolus. The astragalus is sometimes fractured, and the lower extremity of the fibula is broken into several splinters. The internal and external lateral ligaments are usually intact; but if the fibula is not broken, the external lateral ligaments are ruptured. (Note that there is here described the "fracture by adduction" with a splitting off of a greater or less portion of the medial surface of the tibia—illustrated in Fig. 2, from Cooper—a lesion which Tillaux thought had not before his time been observed by any surgeon.)

Under the heading "Fractures of the Tibia and Fibula Near the Ankle-joint" (p. 353), Cooper describes a fracture of the fibula from two to three inches above the ankle-joint (that is, not at a point from two to three inches above the tip of the external malleolus, but at the point where occurs the fracture called by Pott's name), produced by falling laterally while the foot is confined in a deep cleft; and a fracture of the tibia, in which the fracture line runs either obliquely down and in, toward the internal malleolus (*i. e.*, the ordinary spiral fracture of the shaft low down) or obliquely from one or two inches above the internal malleolus down and out into the ankle-joint (*i.e.*, the ordinary splitting fracture of the median part of the articular surface, usually dependent on a primary fracture of the fibula, and forming a less advanced degree of the fracture by adduction already described by Cooper as an outward dislocation of the tibia).

After Cooper. Maisonneuve[4] (1840). In a most remarkable essay, which has been ignored by most subsequent writers, Maisonneuve threw more light on the subject of ankle fractures than has anyone since.

Up to that time, as he says, two theories prevailed to explain the mechanism of fractures of the lower end of the fibula: (1) by adduction, by which means the tip of the external malleolus was torn off; this

was thought by Dupuytren to be the most frequent variety,[22] and (2) by abduction, when, from pressure upward and laterally by the astragalus on the external malleolus, the latter was forced outward until the fibula broke at its weakest point, namely, entirely above the malleolus, that is, in the region described by Pott; this fracture being usually accompanied by a secondarily produced fracture of the internal malleolus or rupture of the internal lateral ligament. The second variety was thought by everybody, except Dupuytren, to be the most frequent.

Now, Maisonneuve proposed another mechanism, which he believed explained the production of the most common type of fracture: this was simple deviation of the point of the foot outward, that is, external rotation of the foot in the tibiofibular mortise around a vertical axis. In one recent necropsy, he had found an oblique fracture of the fibula, the line of fracture beginning on the anterior surface of the external malleolus 4 cm. above its tip and extending upward and backward to a point on the posterior surface of the fibula 8 cm. above the tip of the external malleolus. And he found that precisely this fracture was readily produced in the cadaver by the mechanism of outward rotation as above mentioned, whenever the tibiofibular ligaments held firm.

So far as I can ascertain, Maisonneuve was the first, and I might say almost the only, writer up to the present day to appreciate properly the importance of the inferior tibiofibular ligaments in the mechanism and classification of ankle fractures; and to recognize the great frequency of the oblique fracture of the fibula. This fracture, he found, was always the first lesion following external rotation of the foot, and occurred without any ligamentous injury. If the external rotation of the foot were continued far enough, the internal lateral ligament would rupture, or often the internal malleolus would be pulled off. The resulting deformity, he found, was precisely the picture

[22] Dupuytren contended that the usual fracture which he described (and which he thought corresponded to that shown in Pott's illustration) was produced by a primary tearing off of the external malleolus when the foot turned so that the patient stepped on the outer edge of the sole; and that the rupture of the internal lateral ligament or fracture of the internal malleolus was produced secondarily by the patient's attempts to walk: whereupon, the external malleolus being already broken, the foot was forced into valgus; but he stated that when fracture of the fibula followed a turning outward of the foot, the internal malleolus broke first and the fibula only secondarily (footnote 14, p. 649).

drawn by Dupuytren ("*coup de hache*," etc.); and all of these phenomena disappeared when the position of the foot was corrected. If the inferior tibiofibular ligaments break, during this external rotation of the foot around the long axis of the leg, there occurs a greater or less diastasis of the inferior tibiofibular joint; and if the movement still continues, the fibula breaks not at the usual site, but in its upper third, or at least in its middle third. It is to this fracture of the fibula in its upper third, produced after diastasis of the inferior tibiofibular joint has occurred, that the name of Maisonneuve's fracture has been attached; though he never encountered such a case clinically.

Maisonneuve's chief contribution was his recognition of the oblique fracture of the lower end of the fibula as the first stage of a lesion frequently including also a fracture of the internal malleolus or a rupture of the internal lateral ligament; and it is a pity that his name is not applied to this very frequent fracture rather than to one of the utmost rarity. Moreover, he was no doubt correct, as I have previously intimated, in believing that this was the lesion whose clinical signs were described by Dupuytren, and that the latter erroneously thought these cases were such as had been depicted by Pott. Maisonneuve's chief, and almost his only, error, as it seems to me, was his failure to appreciate that a lesion did exist, consisting of a fracture well above the inferior tibiofibular joint, associated with fracture of the internal malleolus or rupture of the internal ligament, and that this type of fracture, though rare, was yet much less rare than a fracture of the fibula in its upper third or even in its middle third.

After Maisonneuve, Tillaux[3] (1872). Tillaux did not distinguish in his cadaveric experiments between abduction (fibular flexion) of the foot and the same movement combined with slight outward rotation (*i. e.*, around the long axis of the leg). Abduction, he found, caused (*a*) rupture of the internal lateral ligament or tearing off of the internal malleolus, or (*b*) the same lesions plus fracture of the fibula above the inferior tibiofibular ligaments, or 6 to 7 cm. above its tip; the tibiofibular ligaments

may not rupture, but the degree of rupture of these ligaments determined the degree of the displacement; and he found the fibula could not be broken in abduction movements unless the internal malleolus or the internal lateral ligament had been previously broken. He also called particular attention to the great frequency of a fragment of bone torn off the lateral border of the tibia by the inferior tibiofibular ligaments; this fragment has since been known to the French as the "third fragment of Tillaux" (remember it had been described and illustrated by Cooper and by Vidal de Cassis).[23]

It is thus seen that while Maisonneuve minimized the mechanism of pure abduction, Tillaux magnified it out of all reason; for the fact remains that the vast majority of fractures are not of the type produced experimentally by Tillaux by abduction (and which correspond rather to the original type with which Pott and Dupuytren thought they had to deal), but are of the type produced experimentally by Maisonneuve by external rotation. In his description of the results of adduction (tibial flexion), Tillaux agreed with his predecessors and successors (the results are noted below under the account of Hönigschmied's experiments); but he also describes a rare result of adduction which consists in a transverse supramalleolar fracture of the tibia sometimes occurring in those cases in which the fibula

[23] Souligoux (Bull. et mém. Soc. de chir. de Paris, 1912, xxxviii, 1103) described two unpublished diagrams made by Tillaux to illustrate this "third fragment": the anterior view showed the anterior tubercle of the tibia torn off, while the posterior view showed the posterior tubercle torn off. Apparently, then, it was Tillaux's opinion that either tubercle could represent his third fragment. A larger fragment from the inferior lateral margin of the tibia may exist, known to the French as the "intermediate fragment of Verneuil," but it is quite rare. This seems to correspond to what Roberts and Kelly (the advance proof sheets of whose second edition, 1921, Dr. Roberts has very courteously sent me) describe as the "drunkard's fracture." Confusion is added to this matter by the statement of Thaon (Bull. Soc. anat. de Paris, 1870, xlv, 212), in presenting a autopsy specimen of a posterior marginal fracture, that he had often seen this posterior fragment in experiments made by Tillaux; and by the statements of Demoulin (Bull. et mém. Soc. de chir. de Paris, 1912, xxxviii, 1103), of Mauclaire (Bull. et mém. Soc. de chir. de Paris, 1912, xxxviii, 1141), and of Viallet (Rev. de chir., 1912, xlvi, 690) that the third fragment of Tillaux may easily be mistaken in roentgenograms for a posterior marginal fragment. My own belief is that the third fragment of Tillaux is the anterior tibial tubercle and that the fragment composed of the posterior tubercle and that known as the posterior marginal fragment usually are indistinguishable.

breaks above the inferior tibiofibular joint instead of below it (Fig. 43). This mechanism, he says, involves great strain on the superior tibiofibular joint until the fibula breaks above its malleolus; and he observed a case in life in which this transverse supramalleolar fracture (of the tibia only) was complicated by a diastasis of the *upper* tibiofibular joint. (In this connection, one of the specimens from the Mütter Museum[72] is of much interest.)

Hönigschmied[6] (1877), as already remarked, made 125 experiments on the cadaver, to determine the mechanism of ankle fractures, and a brief summary of them is given, as it will be necessary to refer to them frequently in the later portions of this memoir.

1. *Plantar Hyperflexion, Twenty Experiments.* In fourteen cases he got rupture of the internal lateral ligament and anterior fibers of the external lateral ligament; the rupture sometimes occurred from the tarsus, sometimes from the leg bones; sometimes a small fragment of bone was torn loose. In five cases (aged subjects), one or both of the malleoli were broken.

2. *Dorsal Hyperflexion, Twenty-one Experiments,* always with previous division of the tendon of Achilles. In seventeen the internal malleolus was broken, by push of the tarsus against its tip. In two the internal lateral ligament was ruptured. In two the only lesion was in the tarsus.

3. *Tibial Flexion (Adduction or Supination), Seventeen Experiments.* In all the external malleolus (five) or external lateral ligament (twelve) was broken. He never got fracture of the internal malleolus or of the tibia.

4. *Fibular Flexion (Abduction or Pronation), Twenty-two Experiments.* In fifteen the internal lateral ligament or the internal malleolus broke. In seven the only lesions were in the tarsal (two) or subastragalar (five) ligaments. He never obtained a primary fracture of the external malleolus: in two cases only did it break, the lesion being a compression fracture at its tip. [If the inferior tibiofibular ligaments had previously ruptured, the weight of the patient's body, in a fall, would have fractured the fibula above these ligaments].

5. *Inward Rotation of Foot around Long Axis of Leg, Twenty Experiments.* In nineteen there were lesions of the ligaments (fifteen involved the tarsal ligaments, of which six involved also the external lateral

ligament; while in four the external lateral ligament was alone involved). In one the anterior margin of the external malleolus was broken, evidently being torn off by the anterior fibers of the external lateral ligament. (Such a fracture had been described by Wagstaffe,[24] in 1875, and was later studied by L. LeFort[25] (1886) and by his pupil LeRoy[26] (1887).

6. *Outward Rotation of Foot around Long Axis of Leg, Twenty-two Experiments.*

(1) In twenty, fracture of the lower end of the fibula occurred: (*a*) in fourteen of these there was no diastasis of the inferior tibiofibular joint; in two there was rupture of the external lateral ligament with sprain fracture of the tip of the external malleolus. In seven there was only slight obliquity of the fracture line, upward and backward (four had no other lesion, three had also rupture of the internal lateral ligament). In five the line of fracture was distinctly oblique, involving more of the posterior surface of the fibula (two of these five had also rupture of the internal lateral liagment). (*b*) In six cases there was diastasis, usually detaching a small intermediate fragment from the tibia, and with rupture of the internal lateral ligament or fracture of the internal malleolus; in four of these six cases the fracture of the fibula was oblique upward and backward, and in the remaining two cases the fibula was broken obliquely in its upper third (*i. e.*, Maisonneuve's fracture).

(2) In two cases diastasis occurred without fracture of the fibula.

Hönigschmied concluded that these oblique fractures of the lower end of the fibula produced during external rotation are not due, as Maisonneuve taught, to the outward pressure of the astragalus on the anterior border of the external malleolus, but to the pull exerted by the posterior band of the external lateral ligament, which tears off the posterior portion of the malleolus.

Destot, in the thesis of Bondet[27] (1899) and in his own monograph[8] (1911), attempted a physiologic classification, abandoning that based on the mechanism. He pointed out that the main function of the tibia was that of support, while the fibula acted merely as a splint along the outer side of the ankle-joint to maintain the direction of the foot. Thus his classification

[24] Saint Thomas's Hospital Reports, London, 1875, vi, 43.
[25] Bull gén. de Thérap., Paris, 1886, cx, 193.
[26] De la fracture marginale antérieure de la malleole externe. Paris, 1887.
[27] Thèse de Lyon, 1899.

embraces (1) those fractures which involve only the mortise and disturb the equilibrium of the foot; and (2) those which involve the "pilon tibial" (the tibial "pestle") and hence compromise the support of the body. Quénu[28] (1912) addressed very severe criticisms against this classification, and, pointing out that the gravity of the lesions depends entirely on whether they are isolated (involving one bone only) or associated (involving both tibia and fibula), erected on this basis an elaborate scheme which, as I have already said, is less a classification than a catalogue.

Finally, Tanton[29] (1916) attempted to combine the merits of Destot's and of Quénu's classifications, with a reasonable degree of success. Indirect forced movements, he said, may involve (1) the malleolo-astragalar or tarsal ligaments (sprain); (2) the inferior tibiofibular ligaments (diastasis); (3) the malleoli (isolated fractures), very frequent; (4) the "pilon tibial" (isolated), very rare; (5) the malleoli and tibial *pilon* (frequent). Thus he tabulated the lesions as follows (omitting sprains and simple diastases):

I. Fractures of Malleoli—
  1. Isolated:
    (*a*) External malleolus.
    (*b*) Internal malleolus.
  2. Associated:
    (*a*) Low bimalleolar.
    (*b*) Low Dupuytren.
    (*c*) Typical Dupuytren (Pott's).
    (*d*) Maisonneuve.

II. Fractures of Pilon—
  A. Partial.
    1. Isolated:
      (*a*) Anterior or posterior tubercles of tibia.
      (*b*) Marginal: anterior, external, posterior.
      (*c*) Wedge-shaped internal fractures.

[28] Rev. de chir., 1912, xlv, 1, 211, 416, 560.
[29] Fractures du membre inférieur, Paris, 1916.

2. Associated.
   (*a*) Anterior margin with a malleolar fracture.
   (*b*) External margin with a malleolar fracture.
   (*c*) Posterior margin with a malleolar fracture.

B. Total (Eclatements).

Stimson,[7] both in his lecture of 1892, and in the last edition[30] (1917) of his book on *Fractures and Dislocations*, makes a distinction between fractures in which the mechanism of fibular flexion (abduction) predominates and those in which outward deviation of the point of the foot (outward rotation) predominates.

In the former, he says, the internal malleolus is broken, followed by rupture of the inferior tibiofibular ligaments (or sometimes detachment of a piece of the tibia), and then occurs a fracture of the fibula "close above the malleolus"; while in the latter there occurs rupture first of the anterior inferior tibiofibular ligament, then of the anterior fibers of the internal lateral ligament, and almost coincidently the fibula breaks by the twisting of its lower end, the line of fracture being very oblique.

But in spite of all this comparatively explicit and positive teaching for so many generations, the fact is undeniable that the average surgeon knows little or nothing about ankle fractures; and that most teachers of surgery either leave their students with but a confused notion of the subject, or else teach them positively what is certainly not correct.

These examples may be cited:

"Fracture of the lower end of the fibula is a very frequent injury, resulting from indirect violence, the foot, as a rule, being turned violently outward (abduction fracture); as the astragalus forces the external malleolus outward, the tibiofibular ligaments act as a fulcrum, so that the fibula is bent in against the tibia above the attachment of these ligaments, and finally breaks at this point from 5 to 8 cm. above the ankle-joint; the internal malleolus often is avulsed from the tibia, at the same time; and to this combined lesion the

[30] Fractures and Dislocations, ed. 8, Philadelphia, 1917, p. 450.

name of Pott's fracture is given." (Ashhurst: Surgery, Philadelphia, 1914, p. 380.)

"What is a Pott's fracture? . . . It is a fracture of the lower end of the fibula that is produced by the foot turning outward when the injury is sustained. What happens when the foot turns outward? The external surface of the astragalus presses against the tip of the external malleolus, and the fibula breaks primarily. If the interosseous ligament is strong, the fibula breaks within the first two and one-half inches above the tip of the external malleolus, otherwise only the tip of the fibula is broken off on a line with the tibioastragaloid articulation, as in the case before us today. In a typical Pott's fracture the fibula fractures higher up.

"Next occurs a fracture of the internal malleolus, due to traction on the internal lateral ligament or rupture of the ligament. Then, as the pressure is continued with the foot everted and the weight of the patient's body is brought to bear on the foot, what happens? The astragalus is crowded up against the tibiofibular articulation, and, acting as a wedge, forces apart the tibia and fibula and splits or tears the interosseous ligament."[31]

"The mechanism of Pott's fracture consists first in forcible eversion of the foot until the outer surface of the astragalus is driven against the fibular malleolus; secondly, in fracture of the shaft of the fibula, usually above the inferior tibiofibular joint, the unyielding interosseous ligament acting as a fulcrum; thirdly, by a continuation of the vulnerating force, the acute superoexternal border of the astragalus develops as a wedge and is driven upward between the tibia and fibula until it springs the joint by lacerating the interosseous membrane."[32]

Cotton[33] (1910) describes all "eversion fractures" as Pott's fractures, and, though he points out the inaccuracy of this term, does not dwell particularly on their mechanism, merely quoting Stimson's account already given.

Roberts and Kelly[34] (1916), in describing "fracture by eversion and abduction," say the mechanism is first fracture of the internal malleolus or rupture of the internal lateral ligament; "as the force continues the astragalus is forced against the external malleolus, fixing the latter, at the same time the weight of the body falling outward carries with it the leg and the fibula; the fibula is checked by the astragalus [Do they mean that the external malleolus is kept from moving inward as the body of the fibula moves outward?] and

[31] Surgical Clinics of John B. Murphy, Philadelphia, 1914, iii, 2.
[32] Murphy: Surgical Clinics, Philadelphia, 1916, i, 620.
[33] Dislocations and Joint Fractures, Philadelphia, 1910, p. 545.
[34] Treatise on Fractures, Philadelphia, 1916, p. 580.

kept attached to the tibia by the tibiofibular ligament; either the latter ruptures or a line of fracture occurs at the weakest point of the fibula just above the point of attachment of the tibiofibular ligament. It is here that the fixed portion of the fibula meets the potential moving upper portion, and fracture occurs about two to two and one-fourth inches above the malleolus. The line of fracture of the fibula depends to a great extent on whether the fall is directly outward, or whether some torsion of the tibiotarsal joint occurs at the moment of fracture."

On page 590, they describe another (?) mechanism: "In addition to the mechanism described under fibular fracture, there is the effect due to the weight of the body being applied simultaneously with the violence causing the supramalleolar fracture of the fibula; by this force the outer trochlear surface of the astragalus is carried sharply against the outer portion of the articular surface of the tibia by external rotation and abduction of the foot, so that either the tibiofibular ligament must give way or the outer edge of the tibia be broken away from the shaft. Dislocation upward of the astragalus between the tibia and fibula may occur."

Rose and Carless[35] (1920) distinguish between Pott's fracture and Dupuytren's fracture thus:

"In Pott's fracture, sudden abduction, usually combined with eversion of the foot, results in severe strain on the internal lateral ligament, which gives way, or the base of the internal malleolus is torn off. The astragalus is at the same time driven outward against the external malleolus, and the force is thence transferred up the fibula, which bends and breaks at some weak spot. Generally, eversion is a large element in the force that produces the fracture, which then runs obliquely from above downward and forward through the malleolus; less frequently it is due to a pure abduction and may then be situated in the position originally described by Pott, viz., about three inches above the tip of the malleolus, and is transverse, the upper end of the lower fragment being displaced inward toward the tibia. The inferior interosseous ligament remains intact. . . .

"In Dupuytren's fracture a much more serious lesion is produced. The interosseous tibiofibular ligament yields more or less completely, or the flake of the tibia to which it is attached is torn off."

Speed[36] (1916) thus describes the mechanism of fractures about the ankle (p. 772): "When abduction and eversion of the foot are the cause of the fracture, the astragalus is pushed outward, and the

[35] Manual of Surgery, ed. 10, London, 1920, p. 632.
[36] Text-book on Fractures and Dislocations, Philadelphia, 1916, p. 772.

fibula tends to break at a point above the termination [presumably he means the *upper* termination] of the tibiofibular ligament in a transverse or oblique line from compressive force. Coincidently the internal lateral ligament either ruptures, or holding its insertion into the tibia, pulls off the internal malleolus squarely near its lower end (Fig. 566). [This figure represents a "low Dupuytren" fracture.] If this eversion continues strongly, the lower fibular fragment may be separated a little from the tibia by tearing of the tibiofibular ligament, and the internal malleolus is correspondingly dragged outward by the internal lateral ligament, and comes to lie under the joint surface (Fig. 567). [This figure shows a Pott's fracture, with wide diastasis; and the latter certainly preceded the fracture of the fibula and did not, as Speed contends, follow it.] Some torsion is present in all these cases. . . . If the torsion is a more predominating feature in conjunction with the eversion, we obtain the spiral fractures of the external malleolus, as this point projects lower down than the internal malleolus and meets with most of the force in external torsion and eversion. [The reason alleged is insufficient explanation.] These spirals, in a quickly acting force, are above the lower end of the tibiofibular ligament which by a slight elasticity holds while the rigid bone gives; but in slower acting force with more eversion or compressive violence from the body weight, the extreme end of the external malleolus is fractured and splintered up in a spiral manner. As a rule there is not much damage to the internal malleolus and the internal lateral ligament in this mechanism (see Fig. 568)."

This figure shows a low mixed oblique fracture of the fibula, unreduced, though the foot is in adduction and inward rotation. Speed says reduction has not been secured because of laceration of the external lateral ligament. But how could such a laceration occur in the mechanism he is discussing? Speed adds that "sometimes in eversion, in addition to fibular fracture, the tibiofibular ligament is torn, a condition permitting wide separation between the bone ends and possibly accompanied by a shell of bone pulled out from the tibia." Writing (p. 785) of "malleolar fractures caused by inversion of the foot," he says: "The mechanism of isolated fractures of the internal malleolus is that of fall or compression from body weight against the talus, which is tipped inward by the inverted foot. . . . For this result the inversion must not be great, because the pull of the external lateral ligament would also pull off the external malleolus." (Thus he regards fracture of the external malleolus as a secondary and not the primary lesion in adduction fracture. But the illustration he gives as an instance of isolated fracture of the inter-

nal malleolus is not happily chosen [Fig. 592], as it shows the astragalus displaced from the external malleolus, an occurrence obviously permitted only by partial rupture of the anterior fibers of the external lateral ligament or sprain fracture of its tip.)

MECHANISM

Now, after the somewhat tedious historical review given above, it is worth while, before proceeding further in our inquiries, to pause a moment to refresh our knowledge concerning the structure and functions of the ankle. We have read of eversion, of abduction, of rotation, of torsion, compression and bending, of malleolus and fibula, of ligaments and tubercles, of sprains, displacements and diastases; but it is clear that few authors use any of these terms in the same senses, and that some of them probably do not know what they mean themselves. But what Nélaton[37] (1844) said of surgery in general may well be applied to this particular part of surgical knowledge: "*Rien en chirurgie n'est assez abstrait pour que l'obscurité s'excuse par le sujet même, et sur un grand nombre de points la science est assez avancée pour que la verité apparaisse au milieu des controverses à celui qui la cherche sans préoccupation.*" And if what I succeed in explaining in the following pages appears to you to be nothing new, you will admit its truth; and I shall be satisfied. If, on the other hand, you do not agree with my conclusions, that will not in the least disconcert me, for it will not impair their truth.

The ankle-joint is formed above by the tibiofibular mortise, and below by the trochlea of the astragalus, which fits into the mortise as a tenon. This trochlear surface is one-fourth wider in front than behind, conforming to the divergent direction of the internal surfaces of the malleoli. The inferior articular surface of the tibia may be described as the roof or ceiling (*plafond*) of the joint; and the articular surfaces of the malleoli have long been known as the "cheeks" of the mortise.[38]

[37] Eléments de path. chir., Paris, 1844, i, Preface.

[38] From the Greek word for cheek, Quénu derives the adjective *génienne*, which he constantly employs in his classification and nomenclature of ankle fractures, inventing such terms as *bi-malléolaire géni-sus-génienne*, *géni-peronière* and *géni-supramalléolaires*.

That portion of the fibula *which projects beyond the tibial plafond* is properly called the external malleolus; and the corresponding projection of the tibia is called the internal malleolus. The posterior lip of the tibial plafond projects so low as to have been called by Destot the posterior malleolus. This serves to reinforce the mortise posteriorly. It is further deepened by the transverse tibiofibular ligament, which extends from the external malleolus to the posterior lip of the tibia. Thus in walking, as the foot meets the ground in plantar flexion, the leg bones are checked in their tendency to slide forward on the astragalus by the wedge shape of the trochlea of the latter bone (broad anteriorly and narrow posteriorly), by the corresponding divergence of the anteroposterior planes of the malleoli, and by the long posterior lip of the tibial plafond. In walking backward (always digitigrade, not plantigrade) the same mechanism is effective.

The fibula is attached firmly to the tibia, but a slight range of motion is permitted. The interosseous membrane extends throughout the length of the tibial and fibular shafts, the fibers running downward and laterally from tibia to fibula (as in the forearm from radius to ulna); and in addition, there are strong ligaments whose fibers run in the same direction, binding both ends of the fibula to the tibia, at which points only are the two bones in contact. At the upper end, where the head of the fibula butts against the overhanging external condyle of the tibia, there is a synovial cavity to the tibiofibular joint; but at the lower end, where the fibula is received into a longitudinal groove between the anterior and posterior tubercles on the lateral surface of the tibia (Fig. 4), no such joint cavity exists, union being effected by a dense feltlike interosseous ligament, reinforced anteriorly by the anterior inferior tibiofibular ligament, and posteriorly by the posterior ligament of the same name. The malleoli, of which the external is the longer and is situated more posteriorly, serve to keep the foot (which is appended to the astragalus) under the leg bones. The astragalus itself has no muscles attached to it, and serves only as a ball

in a ball-bearing joint to facilitate movements of the leg bones above it and of the tarsal bones below and in front. The foot is attached to the leg bones by ligaments, of which the lateral portions are best developed, constituting for the ankle, as in

FIG. 4.—Transverse section of leg through inferior tibiofibular joint, showing fibula lodged in its groove formed by the anterior and posterior tubercles of the tibia. Note the obliquity of the intermalleolar axis, forming an angle of 30 degrees with the axis of motion of the ankle-joint, which is nearly transverse. Note that the anterior tubercle projects laterally much farther than the posterior, so that in anteroposterior roentgenograms its shadow much overlaps that of the fibula. (From a preparation in the laboratory of operative surgery, University of Pennsylvania.)

other hinge joints, lateral ligaments which hinder motion except in the anteroposterior plane. The internal lateral ligament passes from the internal malleolus in radiating direction (1) anteriorly to the scaphoid and to the median surface of the calca-

neum (sustentaculum tali) and (2) posteriorly to the median tubercle on the posterior surface of the astragalus. The external lateral ligament has three distinct bands, passing from the external malleolus: one goes forward to the lateral border of

FIG. 5.—Posterior view of the ankle-joint, all structures removed except the bandlike ligaments. Note the interosseous membrane, its fibers passing downward from the tibia to the fibula, the similarly directed fibers of the posterior inferior tibiofibular ligament; the middle band of the external lateral (fibulocalcanean) ligament; the posterior band of the external lateral ligament (fibuloastragalar ligament) attached to the lateral tubercle of the astragalus; the posterior fibers of the internal lateral ligament, and the posterior surface of the internal malleolus grooved for the tendon of the tibialis posticus. (From a preparation in the laboratory of operative surgery, University of Pennsylvania.)

the neck of the astragalus, just above the sinus of the tarsus; the middle band passes downward and slightly backward to the calcaneum; while the posterior, whose deep portion is extremely strong (Fig. 5), is attached to the lateral tubercle

on the posterior surface of the astragalus (os trigonum), which, being from 5 to 7 mm. posterior to the median tubercle, is the portion of the astragalus which casts the farthest posterior shadow in lateral roentgenograms of the foot. This posterior band of the external lateral ligament is so exceedingly strong that it is very seldom ruptured; it holds the astragalus almost indissolubly attached to the external malleolus,[39] and in injuries of the ankle either one or other bone to which the ligament is attached is more easily broken than is the ligament ruptured. As will be shown subsequently, usually the fibula gives way when the strain comes; occasionally, however, the posterior tubercle of the astragalus is detached; and I have seen at least one case in which fracture occurred at both points simultaneously.[40]

The next question that arises is, What is the function of the fibula?

Humphry[41] (1858) notes that the fibula is an inconstant bone in animals: in carnivora and pachydermata it extends from the upper end of the tibia to the ankle, as in man. In most rodents it is united with the tibia at the lower part. In ruminants it altogether disappears. In birds its upper extremity enters into the knee-joint and articulates with the external condyle of the femur; it lies close against the tibia and dwindles and disappears about the middle of the leg. In reptiles it is large, in many extending to the knee-joint above and to the tarsus below. In the bat the lower half of the fibula and the upper half of the ulna are retained.

It has seemed to me, from study of the skeletons in the Museum of the Academy of Natural Sciences of Philadelphia, that the fibula was best developed and extended farthest beyond the tibia at the ankle in those animals which were most nearly plantigrade, and in which, as in man, stability rather than agility was demanded. In digitigrades, such as the horse and camel, there is no fibula; in partial or less complete digitigrades (rhinoceros) it extends beyond the tibial

[39] Chaput (Bull. et mém. Soc. de chir. de Paris, 1912, xxxviii, 1192), says they are like Siamese twins.

[40] The posterior tubercle of the astragalus may sometimes be fractured by direct violence as it is crushed against the posterior lip of the tibia in forced plantar flexion of the foot.

[41] Treatise on the Human Skeleton, Cambridge, 1858, p. 490.

plafond, but not below the level of the internal malleolus. In a still less complete digitigrade (almost a plantigrade), such as the elephant, it extends below the level of the internal malleolus, and there is outward rotation of the lower end of the tibia, as in man. In the gorilla, chimpanzee, orang-utan, etc., on the other hand, which are more plantigrade than digitigrade (but in which, as already noted, agility is retained at the expense of stability) the lower end of the tibia has not rotated out as far as in man (in fact, not so far out as a transverse plane through the tibial condyles), and the fibula descends no lower than the internal malleolus. This lack of development of the fibula in these more or less anthropoid animals was noted by Bland-Sutton[42] (1888); and he further calls attention to the fact that in babies born with congenital clubfeet this deformity is merely a lack of normal development, the outward rotation of the lower end of the tibia not having occurred, and the external malleolus not having descended below the level of the internal. Evidently, he concluded, the external malleolus was developed only to aid members of the human race to walk in the erect posture; it was required to keep the foot steady and prevent it from turning outward into a position of extreme valgus. As Destot says, the external malleolus acts merely as a splint to maintain the direction of the foot.

But if this is so, why is the fibula a separate bone in man? Why is not the external malleolus merely a part of the tibia? To this I know of no better answer than that given by Bromfeild[11] (1773) that if it were a part of the tibia, and no give or play whatever occurred between the malleoli, no one could take more than a very few steps without fracturing one or other malleolus.

If a leg is studied from which all soft parts have been removed except the ligaments and the interosseous membrane, these phenomena may be observed:

In full plantar flexion (extension) of the foot, the anterior and middle bands of the external lateral ligament become tense and pull the external malleolus medially (and slightly downward and backward) against the tibia, keeping the external malleolus in close contact with the astragalus as this glides forward and presents to the intermalleolar space a slightly less diameter than in full dorsiflexion of the foot. In the latter movement, dorsiflexion, the anterior band of the external lateral ligament becomes lax, and as the external malleolus is forced away from the tibia and slightly upward, the anterior and posterior tibiofibular ligaments become tense, especially the anterior ligament; also, as flexion beyond a right angle occurs,

[42] Am. Jour. Med. Sci., 1888, xcv, 376.

much tension develops on the middle band of the external lateral ligament, and this pulls the malleolus backward: the posterior band of the external lateral ligament is always tense; it makes the astragalus and external malleolus practically one bone.

The expansion of the intermalleolar space which occurs during dorsiflexion of the foot may amount to several millimeters. I have measured it by affixing a wire in each malleolus and bending the ends of these wires forward over the ankle-joint until they crossed each other in parallel lines: by scratching a mark on each wire at the same point when the foot is in full plantar flexion, it is easy to measure the excursion as the foot is brought up into full dorsiflexion. This excursion, which exceeds 2 mm. and may approach 3 mm., allows the wider anterior diameter of the articular surface of the astragalus to pass back between the malleoli in dorsiflexion, while the downward drag of the anterior and middle bands of the external lateral ligament keeps the malleoli in contact with the small posterior diameter of the astragalus which presents between them in full plantar flexion. Destot[8] (1911) pointed out that inasmuch as the intermalleolar axis and the axis of rotation of the astragalus, in flexion and extension, do not coincide (they form an angle of about 30 degrees, open laterally, Fig. 4), it is not a directly transverse diameter of the astragalus that presents between the malleoli at any point of flexion or extension, but a diameter of varying obliquity which is, however, always nearly the same in length; and he is inclined to ignore the existence of the movements of the external malleolus. And I have dwelt at some length on these movements because I have found them nowhere described and because there has been some dispute about them. Humphry[41] (*loc. cit.*, p. 557) asserted that the increase of the distance between the malleoli was secured solely by the elasticity of the fibula, which bent inward toward the tibia in its lower fourth, when the external malleolus was forced outward. Nancrede[43] (1880), however, pointed out that an upward and downward movement of the fibula occurred, and asserted that Humphry's theory was "preposterous and untrue."[44] This is my own opinion also.

The upper end of the fibula can also be seen to move in flexion and extension of the foot at the ankle; in full plantar flexion (with median and downward movement of the external malleolus), the superior end of the fibula moves slightly forward and rotates slightly outward, its anterior surface turning away from the tibia. This movement is due largely to the median and backward pull exerted on the external

[43] Phila. Med. Times, 1880, x, 316.
[44] Nancrede: Maryland Med. Jour., 1880, vii, 76.

malleolus by the middle and posterior bands of the external lateral ligament. The head of the fibula slides backward and very slightly upward again in full dorsiflexion. Thus the chief movement of the superior tibiofibular joint is an anteroposterior one (downward and forward, or upward and backward) around the inferior tibiofibular joint as a pivot; but these movements are so slight as to be scarcely appreciable.

Very little change occurs in the interosseous membrane during these movements, except in its lower fourth, where it spreads and becomes tense (the aperture for the anterior peroneal artery tends to become round from oval) as the external malleolus ascends and moves backward; and it again becomes relaxed when the external malleolus descends and moves mesially toward the tibia. The strength of the interosseous membrane is much greater than usually supposed. Even when the lower end of the fibula is freed from its tibial attachments, very great force is required to rupture the interosseous membrane, and fracture of the fibula in its lower third is the nearly invariable sequel.

The normal movements of the ankle-joint are those of flexion and extension—20 degrees of dorsiflexion and 60 degrees extension or plantar flexion, a total of 80 degrees approximately. This motion occurs around an axis which passes in the frontal plane somewhat below and in front of the tip of the external malleolus. This axis makes an angle of 30 degrees (thereabouts) with the bimalleolar axis. This arrangement accounts for the greater excursion forward of the lateral astragalar surface in relation to the external malleolus as compared with the motion which occurs between the median surface of the astragalus and the internal malleolus; as well as for the apparent deviation of the point of the foot medially in full plantar flexion, and laterally in full dorsiflexion.

If movements were possible in the ankle-joint around an anteroposterior axis, they should be named adduction (tibial flexion) and abduction (fibular flexion), or movements in the frontal plane toward and away from the median line. These movements are quickly resisted by the tension on the lateral ligaments (Figs. 6 and 7), and if forced, the malleolus *away from which motion occurs* is torn off by its ligament, or the liga-

ment itself ruptures (see the experiments of Hönigschmied, related at page 658). Motions of adduction and abduction in the foot normally occur in the subastragalar joint and permit the calcaneum without difficulty to accommodate itself to slight irregularities of the soil. But the calcaneum is attached to the astragalus by the extremely strong astragalocalcanean

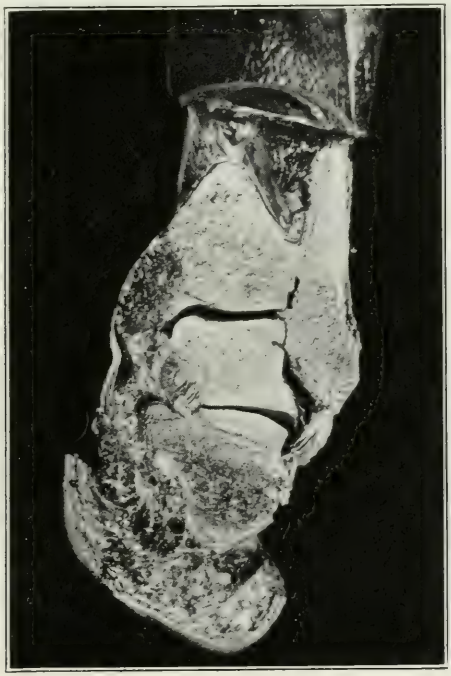


FIG. 6.—Frontal section of the ankle-joint. Adduction causes tension on external lateral ligament and if forced will tear off the external malleolus.

interosseous ligament; and when such movements are too extensive, they are transmitted directly to the astragalus and from it to the tibiofibular mortise where fracture of one or the other malleolus is the usual consequence. Not until that malleolus on which the pull comes has given way, or the corresponding ligament has ruptured, is the astragalus able to act on the other malleolus by a push so as to produce a compression

fracture. Rare exceptions to this general rule occur, however, as when the calcaneum is itself first broken, and becomes so displaced as to press directly on the end of the fibula, producing a compression fracture of the external malleolus (Fig. 23).

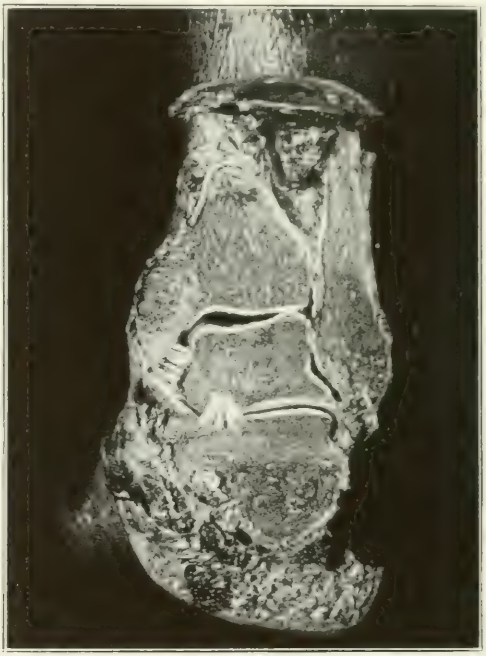


FIG. 7.—Frontal section of the ankle-joint. Abduction causes tension on internal lateral ligament and will rupture this or fracture the internal malleolus before outward pressure on the external malleolus will fracture the latter or cause diastasis between tibia and fibula. (This specimen and that shown in Fig. 6 are from a preparation in the laboratory of operative surgery, University of Pennsylvania.)

Movements of rotation around the long axis of the leg may be attempted in the ankle-joint by twisting the point of the foot toward or away from the median line. As pointed out by Maisonneuve[4] (1840), movements of inward rotation are almost inseparable from a movement of adduction, as the numerous joints in the anterior tarsus render the foot very mobile in this

direction. Any movement toward outward rotation, however, converts the foot into a rigid lever, and motion is easily and with much force transmitted to the ankle-joint, the astragalus attempting to turn so as to bring its long axis crosswise between the malleoli. Owing to physical laws, it is on the external malleolus that the greatest strain comes. Maisonneuve illus-

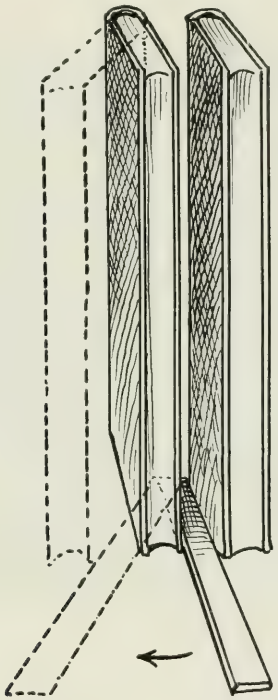


FIG. 8.—An illustration of Maisonneuve's explanation of fracture of the external malleolus by outward rotation of the foot around the axis of the leg. The books represent the malleoli and the ruler represents the foot. See page 675.

trated this by placing a ruler (which represents the foot) between two parallel volumes (which represent the malleoli): the volume which is moved is always that toward which the long end of the lever moves (Fig. 8); even if this volume be much heavier than the other it is easily moved by the greater leverage exerted. In movements of outward rotation, the foot, relatively to the

tibia, is a lever of the first order, with its fulcrum on the anterior border of the fibula: the arm of the resistance will have, say, a length of 3 cm., that of the power 12 cm. (the length of the foot being taken as 15 cm. from the toes to the posterior border of the ankle-joint). Relatively to the fibula, it is a lever of the second order, with its fulcrum at the posterior border of the internal malleolus: the resistance, therefore, has an arm of 3

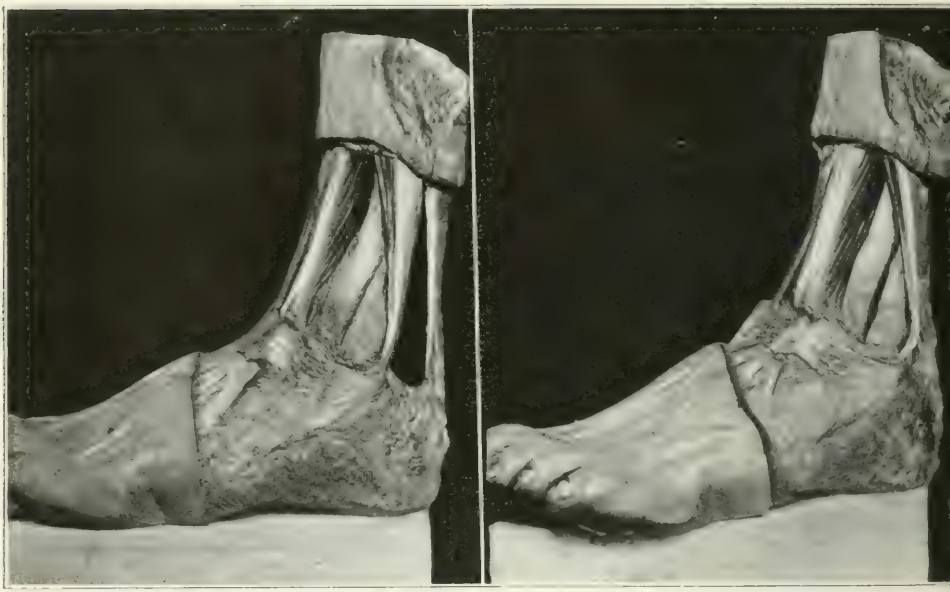


FIG. 9.—Oblique fracture of the fibula through the inferior tibiofibular joint, produced by an osteotome: in the first figure the foot is in the anatomic position; in the second it has been rotated outward around the axis of the leg, which is the only motion that will cause separation of the fragments. See page 676.

cm. (as in the other case), but the power has an arm of 15 cm. (the whole length from the point of the foot to the fulcrum). Thus the force which tends to fracture the fibula is as 12 is to 9, or as 4 is to 3. Such a mechanism as this (outward rotation of the foot) usually causes an oblique fracture of the lower end of the fibula (see Hönigschmied's[56] experiments, page 658); and if such a fracture be made by an osteotome it will be found that

external rotation is the only movement that will cause separation of the fragments (Fig. 9). It is evident that the mechanism of this fracture, which is the most frequent of all fractures of the ankle (more than 25 per cent of all cases), involves not only a push outward on the anterior border of the external malleolus, as noted by Maisonneuve, but also, as Hönigschmied pointed out, a pull inward on its posterior border by means of the posterior band of the external lateral ligament. The line of this fracture is oblique from above and behind, downward and forward. It is, properly speaking, a spiral fracture produced by torsion. Its obliquity varies greatly; but it is always higher on the posterior surface of the fibula than on its anterior, and the line of fracture passes through and involves the inferior tibiofibular joint. Almost invariably, its lower and anterior end extends to the external malleolus (in 90 per cent of our cases): often just below the tibial plafond, sometimes as far down as the very tip of the malleolus. Thus in practically every instance the anterior inferior tibiofibular ligament remains intact, or even if partially ruptured, there results no true diastasis between fibula and tibia. At most, the lower fragment, comprising that part of the fibula posterior to the attachment of the anterior tibiofibular ligament, rolls outward and slightly backward around the unruptured posterior tibiofibular ligament as a hinge.

If this "mixed oblique"[45] fracture of the fibula, as Destot names it, is the sole lesion resulting from outward rotation of the foot, there is little or no displacement (Figs. 10 and 11). This was the case in all of the seventy-nine cases studied by Dr. Bromer and myself. If the force continues to act, the next lesion which is added is rupture of the internal lateral ligament (in twelve cases only, in our series), or, far more frequently, fracture of the internal malleolus, usually only of its anterior tip, seldom of its whole extent (Fig. 12). This combined lesion (oblique fracture of the fibula with fracture of the internal malleolus) occurred in thirty-two cases in our series, or in 10

[45] "Mixed" because involving the fibula both above and below the tibiofibular joint.

per cent of the entire number. The displacement may be slight or marked. And in very many cases (fifty-one additional cases in our series), besides these two lesions, there is added the complication of fracture of the posterior margin of the tibia. Counting in all complications and variations, this type of fracture

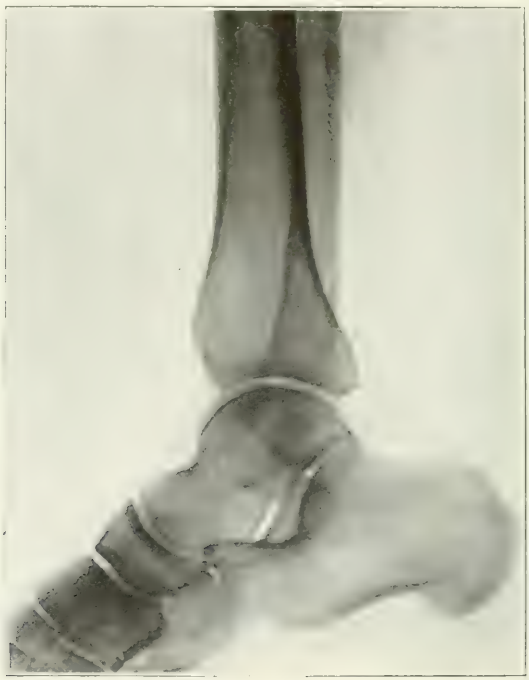


FIG. 10.—Mixed oblique fracture of the lower end of the fibula: the first lesion resulting from external rotation (A, I); occurs as isolated lesion in 25 per cent of all cases of fracture of the ankle; as combined lesion, in 61 per cent. Usually invisible in anteroposterior views.

occurred in 100 cases or in 33 per cent of the total 300 cases we have studied.[46]

Seldom does the obliquity of the fibular fracture pass so high

[46] Chaput (Bull. et mém. Soc. de chir. de Paris, 1906, xxxii, 1047) noted that among 130 cases of fracture at the ankle, of which he had studied the roentgenograms, 113 conformed to this oblique type of fracture of the fibula, against only 17 cases in which the fracture was clearly above the malleolus.

as to be above the level of the anterior inferior tubercle of the tibia (Figs. 13 and 14): in three cases this tubercle was de-

FIG. 11.—Mixed oblique fracture of the fibula, with great obliquity. In these fractures the obliquity may be great or slight; but the line of fracture is always higher on the posterior than on the anterior border of the fibula, and in 84 per cent of the cases the anterior end of the fracture line is between the tip of the malleolus below and the level of the articular surface of the tibia above. In 8 per cent of the cases the anterior end of the fracture line passes through the fibula at the level of the anterior tubercle of the tibia, and in 8 per cent its anterior end is above this level (in which circumstances the tubercle is detached or there is diastasis). All tracings here reproduced were made directly from the roentgenograms.

tached (in two of these there was no appreciable lesion at the internal malleolus, but in one the internal malleolus was fractured), and in two others it is probable that a disjunction of the

FIG. 12.—Mixed oblique fracture of fibula, with fracture of internal malleolus (A, II b). The fibula fracture is visible only in the anteroposterior view (rare). Note that there is considerable lateral displacement of the astragalus, but that the posterior displacement is only apparent, not real, being due to the external rotation of the foot.

joint had occurred, as indicated by suggestive roentgen-ray findings. This lesion (fracture of the anterior tibial tubercle, or diastasis of the tibiofibular joint) must occur previous to, even if nearly simultaneously with, the oblique fracture of the

fibula;[47] because after the fibula is fractured its lower fragment (on which alone the force is acting) is already detached from the anterior tubercle of the tibia, and a continuance of the force would merely increase the separation. Moreover, there is lack of displacement in these isolated oblique fractures of the fibula merely because to permit displacement it is necessary

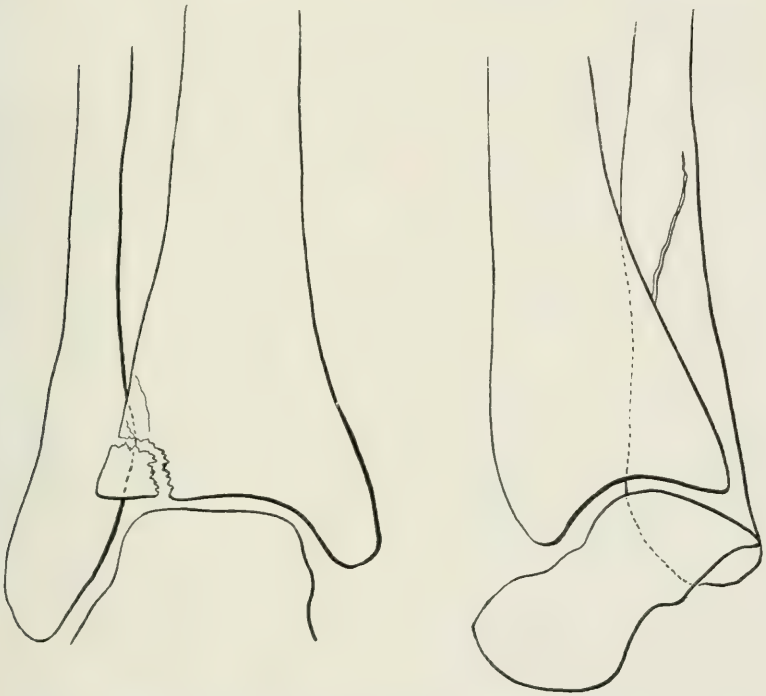


FIG. 13.—Mixed oblique (incomplete) fracture of fibula, complicated by, and subsequent to, avulsion of the anterior tubercle of tibia.

that the internal malleolus (or its ligament) previously give way. If a tibiofibular diastasis (with or without separation of the tubercle) occurs (and if it occurs it must always occur

[47] Lapointe (Souligoux: Bull. et mém. Soc. de chir. de Paris, 1914, xliv, 1042) quite justly contends that if fracture of the anterior tubercle can occur as an isolated lesion (as in the case he reported), it must be admitted it may also occur as the first lesion of a more complicated fracture.

previous to a fracture of the fibula, as already remarked), then the fracture of the fibula (by torsion still) occurs not through the inferior tibiofibular joint but above it, sometimes through its surgical neck, often through its true neck in the upper third

FIG. 14.—Mixed oblique (incomplete) fracture of fibula complicated by, and subsequent to, avulsion of anterior tubercle of tibia.

of the fibula, as originally pointed out by Maisonneuve. That such a lesion in the upper third of the fibula can occur without appreciable bony lesion at the ankle cannot be denied. We have two such cases; and Quénu[48] went so far as to say that

[48] Rev. de chir., 1907, xxxv, 897.

many more such fractures occur without diastasis than with it; and that they may occur even without any lesion at all at the ankle-joint (Fig. 15).[49] It must also be recognized that diastasis is not necessarily followed by fracture of the fibula at

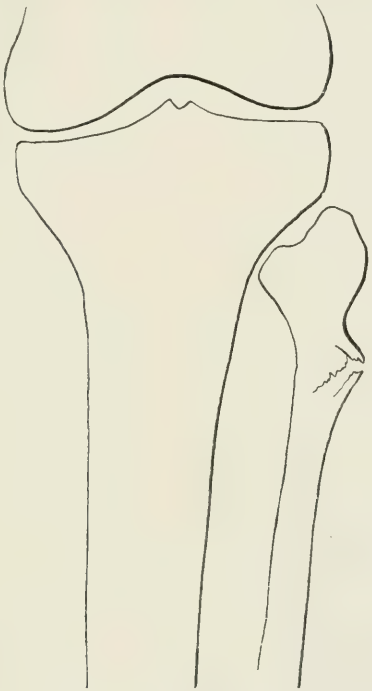


FIG. 15.—Incomplete fracture of fibula below its head (A, I, Variant), apparently from compression in its long axis (see pp. 682 and 695). No bone lesion at ankle. There was a history of a fall down three steps while carrying a load of 100 kilograms on the back. Injury occurred so quickly the patient did not know whether he turned his ankle or struck the side of his leg against the marble steps. Pain was so great that he had to be brought to the hospital by a patrol wagon. Examination showed fracture in the upper third of the left fibula (crepitus and deformity). The ankle was swollen over the external malleolus.

any level, as shown in the lesion represented in Figs. 16 and 17, in which the fibula detached the anterior inferior tubercle of the tibia, and in which, after rupture of the internal lateral

[49] Long recognized as possibly due to pull of the biceps muscle. See a study by Lonhard (Deutsche mil.-ärztl. Ztschr., 1914, xliii, 219).

ligament had occurred, the fibula was forced by the astragalus around back of the tibia by continuance of external rotation of the foot, as in the mechanism described by Huguier[50] (1848),

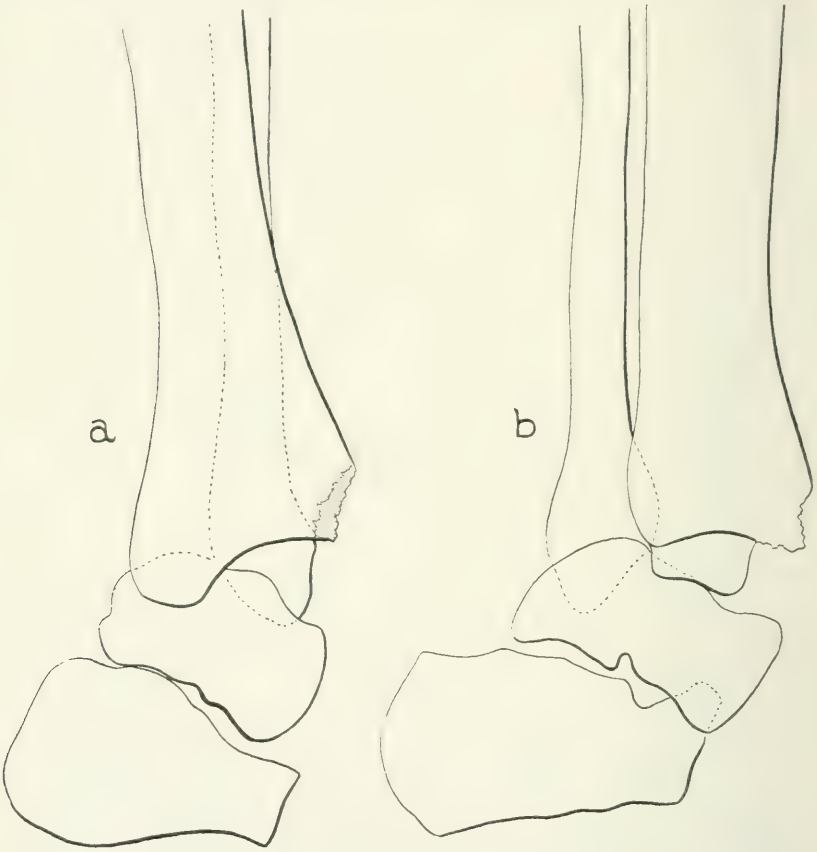


FIG. 16.—Diastasis with displacement of fibula behind tibia. The anterior tubercle of tibia has been torn off and accompanies the fibula, which is not fractured. Diastasis by external rotation (mechanism described by Huguier[50]): (*a*) antero-posterior view, and (*b*) lateral view before reduction. See also Fig. 17 (A, I, complication) and page 683.

though in his cadaveric experiments, as well as in the case illustrated by Destot (Fig. 67, p. 142 of his monograph), this

[50] Union méd., Paris, 1848, ii, 120.

displacement was accompanied by (and I believe succeeded by) a fracture of the upper end of the fibula.

In rare instances, the avulsing force on the internal malleolus may be so great as to cause fracture of the entire lower end of

FIG. 17.—Same case as that shown in Fig. 16, after reduction. Fracture of anterior tubercle of tibia clearly seen.

the tibia (or in children a separation of the epiphysis[51]): this appears to have been the mechanism in four of our cases, which

[51] In addition to the 300 cases of fracture of the ankle listed on page 725, we have records of at least nine cases diagnosed as epiphyseal separations; but as in these the roentgenograms available showed no gross separation at the epiphyseal line of the tibia, we have not included them in our statistics.

on a purely anatomical classification should perhaps be grouped with the supramalleolar fractures (Figs. 18 and 19).

The significance of the posterior marginal fragment, and the mechanism by which it probably is produced, will be discussed in another place (p. 702).

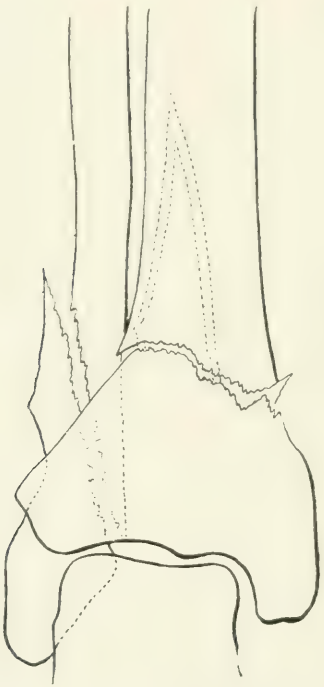


FIG. 18.—Mixed oblique fracture of fibula, internal malleolus represented by whole lower end of tibia (A, III).

I desire to return now to the movement of *forced abduction*, a discussion of which has been intentionally postponed until disposition had been made of the much more frequent mechanism (outward rotation). We find in any large series of fractures at the ankle a certain number in which fracture of the internal malleolus is the only lesion (in our series this lesion occurred in more than 6.5 per cent of the whole number). Now it is not

rational to suppose that the same mechanism which at one time causes an isolated oblique fracture of the lower end of the fibula

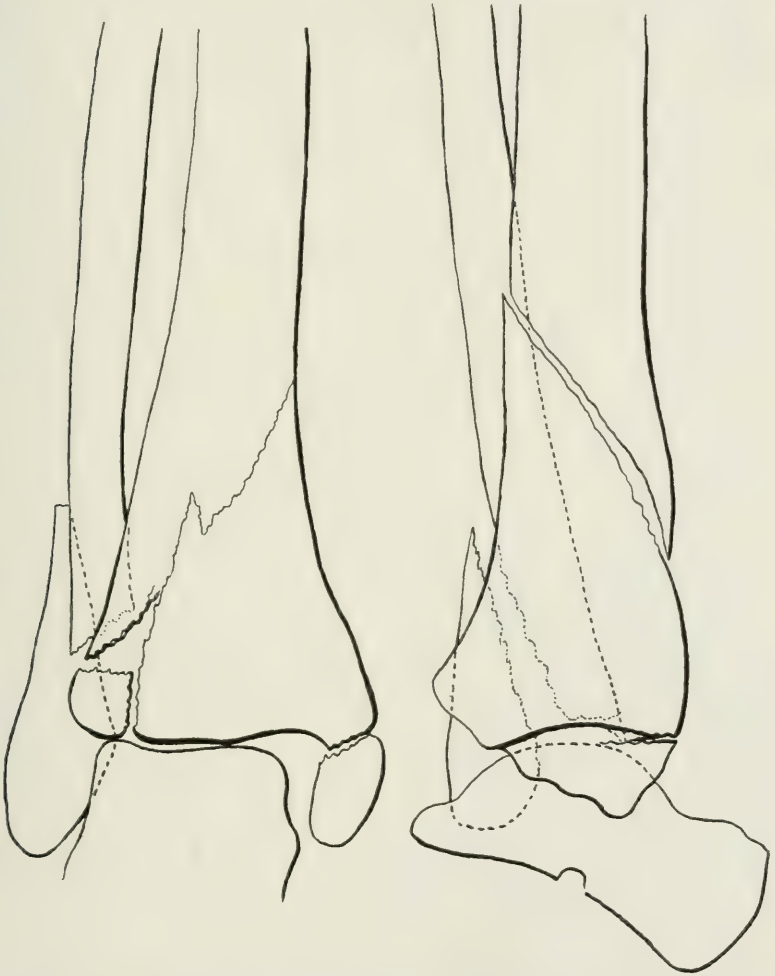


FIG. 19.—Mixed oblique fracture of fibula, with fracture of internal malleolus and of whole lower end of tibia. There is also a fracture of the anterior tubercle of the tibia (A, III).

will at another cause an isolated fracture of the internal malleolus: they must be produced by different mechanisms. Experi-

mentally, it is very clearly seen (note the experiments of Hönigschmied, detailed at page 658, that straight abduction (fibular flexion) of the foot has as its primary and most constant lesion fracture of the internal malleolus, or its equivalent, rupture of the internal lateral ligament. This is a prerequisite in order

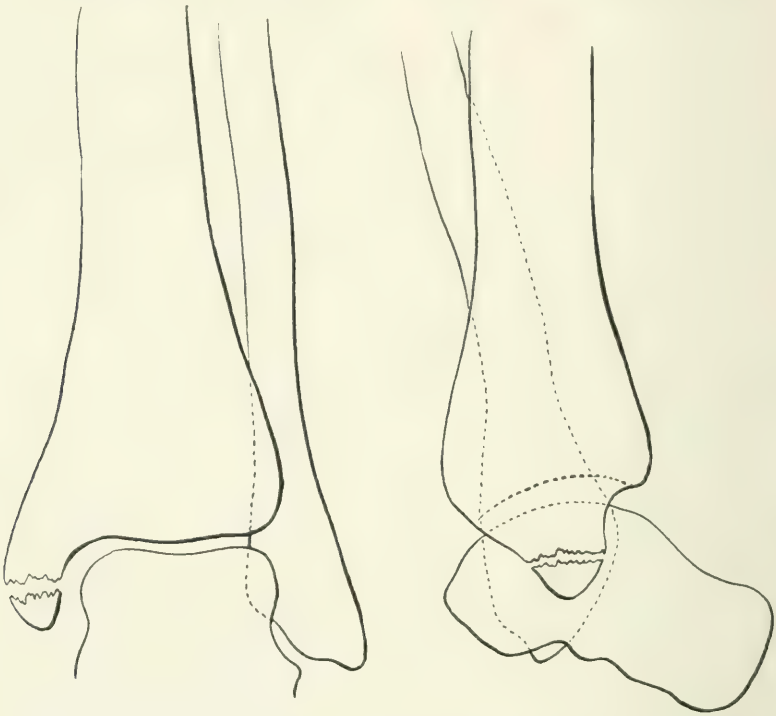


FIG. 20.—Abduction fracture, first degree (B, I): avulsion of internal malleolus. In this patient the internal malleolus extended abnormally low. This fracture occurs in 6.6 per cent of all fractures at the ankle. All forms of the abduction type together comprise about 21 per cent of the cases.

to free the astragalus sufficiently so that it may press directly on the external malleolus, which it does by rotation around an anteroposterior axis. The experiments of Bonnet[52] (1845), have been overlooked by most students. He showed, long

[52] Traité des maladies des articulations, Lyon, 1845, ii, 428.

before Tillaux or Hönigschmied, that abduction (fibular flexion) of the leg, while the foot was held in a vise, caused: first, fracture of the internal malleolus or rupture of the internal lateral liga-

FIG. 21.—Fracture of fibula below inferior tibiofibular ligaments, with very slight obliquity, accompanied by rupture of the internal lateral ligament, as evidenced by lateral displacement of astragalus. This type of fracture is intermediate between the fracture by external rotation already discussed and the fracture by abduction. It is impossible from the roentgenogram alone to say to which of the two types such a fracture really belongs (A, II or B, II).

ment (Fig. 20), and, if the abduction was increased, a crushing or fracture of the external malleolus (Figs. 21, 22), never of the fibula above the inferior tibiofibular joint. But sometimes no lesion of the external malleolus was caused even when the in-

ternal malleolus was widely separated. If, on the other hand, abduction of the foot was produced with the leg lying on its fibular side, but with the foot projecting free of the table, the

FIG. 22 (B, II, a).—Fracture of both malleoli by abduction. A heavy steel I-beam; lying on the ground beside the patient while he was standing at work was tipped over and struck the lateral surface of his leg, causing sudden abduction of the foot. He was not knocked down, but he felt a sudden pain in the ankle. He was able to walk on the leg. In the lateral view, the fracture of the external malleolus (which occurred subsequent to that of the internal malleolus, and was incomplete and subperiosteal) is not visible at all, while even that of the internal malleolus can hardly be seen.

same lesions occurred at the internal malleolus; but the fibula broke above the inferior tibiofibular ligaments at the point where it rested on the table.

In the mechanism of these fractures by abduction the influence of the tibiofibular ligaments is paramount:

1. If the tibiofibular ligaments hold, the fibula breaks across through the external malleolus proper (*i. e.*, below the tibiofibular ligaments) and not above these ligaments by that "preposterous and untrue" mechanism to which Nancrede objected,

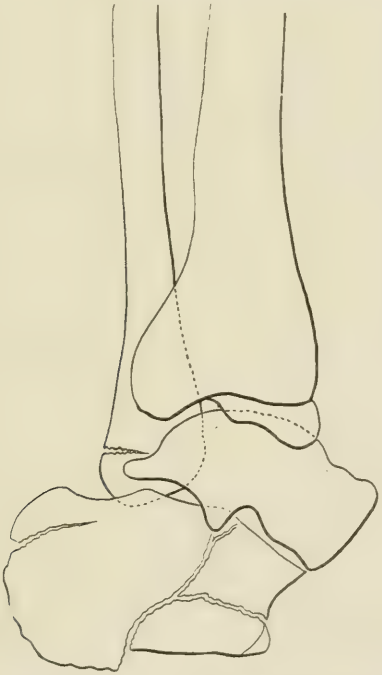


FIG. 23.—Crush fracture of calcaneum permitting fracture of external malleolus by compression. See page 674.

namely, the inward bending of the fibula toward the tibia. It is not proper, perhaps, to deny that the latter mechanism might sometimes occur (all things are possible) in a patient with exceedingly relaxed ligaments; but I feel strongly inclined to state in the words of Souligoux (applied by him to the existence of an isolated fracture of the posterior tibial margin) that I do not believe such a mechanism exists, and will not believe it

exists until somebody shows me its method of production. Of the 300 fractures which we have studied, we find only thirteen cases which seem to belong to this type (bimalleolar fracture by abduction): evidently the more nearly the movement of the foot conforms to the type of straight abduction, the more apt

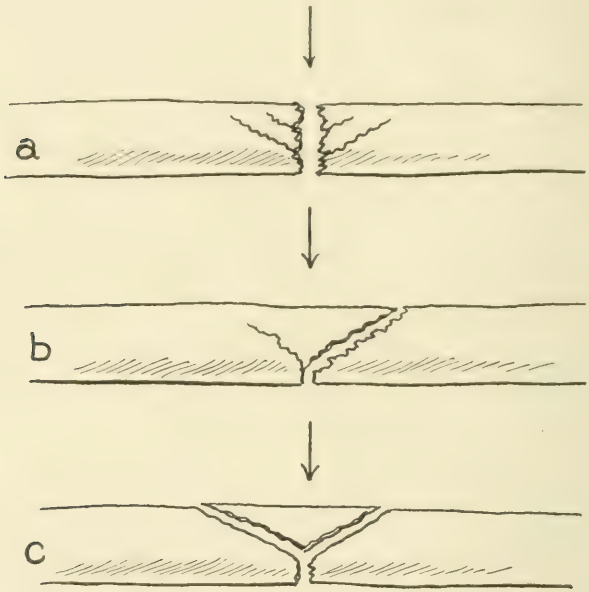


FIG. 24.—Messerer's diagram illustrating the mechanism of fracture by bending (Biegungsbruch) or flexion: (*a*) minute examination always shows in fractures apparently transverse delicate lines diverging from the main fracture and extending backward toward the surface of the bone which has been compressed or flexed, and away from the surface of extension; (*b*) an oblique fracture represents one of these diverging lines which has become a complete fracture; (*c*) very frequently a wedge-shaped fragment is detached, with its base on the compression (concave) surface and its apex toward that of extension (convex surface). See Figs. 25, 26, 28, 29, 35, 36.

is diastasis, and as a consequence, fracture above the tibiofibular ligaments to occur (see below); while the more nearly it corresponds to external rotation (deviation of the point of the foot outward), the more certain is the fracture to be oblique in type, involving the inferior tibiofibular joint, but, as already

explained, causing no true diastasis; hence the extreme rarity of true bimalleolar fractures by abduction (Fig. 22).[53]

2. If the tibiofibular ligaments rupture, then the fibula is freed from the tibia, and if the force continues (in life it is now

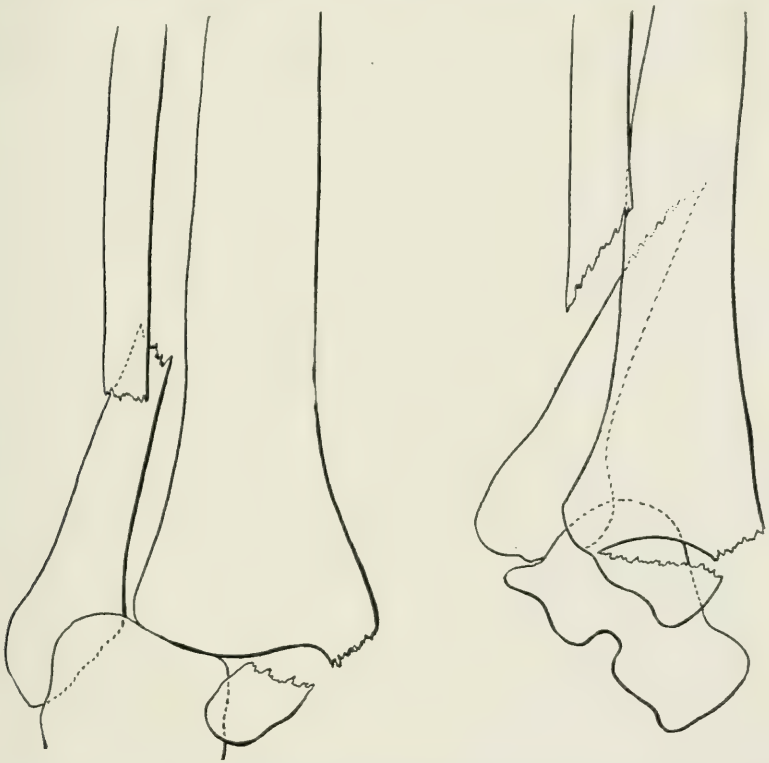


FIG. 25.—Fracture by abduction, second degree (B, II, b) (Pott's fracture, Dupuytren type): a primary fracture of the internal malleolus or rupture of the internal lateral ligament, followed by diastasis (with or without an intermediate fragment), finally succeeded by fracture of the fibula above the inferior tibiofibular joint by bending (Biegungsbruch).

the weight of the body borne chiefly, or at least abnormally, on the fibula), the fibula breaks "by flexion" and the break usually occurs where the fibula is weakest, through the surgical

[53] Mention has already been made (p. 674) of isolated fracture of the external malleolus associated with fracture of the calcaneum (Fig. 23).

neck above the inferior tibiofibular ligaments. Study of roentgenograms or museum specimens of fractures of this type shows clearly two things: an evident tibiofibular diastasis, and a flexion fracture (Biegungsbruch) of the fibula. The typical mechanism of the flexion fracture is illustrated in Fig. 24

FIG. 26.—Fracture by abduction, second degree (B, II, b) (Pott's fracture, Dupuytren type): diastasis evidenced by detachment of fragment from anterior tubercle of tibia (intermediate fragment); fracture by bending clearly shown by detachment of wedge from concave side of fibula.

(p. 692), copied from Messerer; and though at first glance some roentgenograms of fractures of the fibula at this height may not seem to indicate this mechanism clearly, more careful study (in nearly every case, at least, with which I am familiar) shows that the fracture conforms to the Biegungsbruch type: thus the line of fracture is either nearly transverse (rare), slightly

oblique (frequent, Fig. 25), or (very frequent) is comminuted in the typical manner, with detachment of a wedge-shaped fragment from the concavity of the bent bone (Figs. 26 and 35); that is to say, the wedge is on the lateral border of the fibula (apex toward the tibia) if the fibula was broken by straight abduction of its lower end, or on the posterior border (apex anteriorly) if it broke by posterior displacement of its lower end (Figs. 35 and 36).

Now it is a prerequisite for a bone (or any other similarly shaped structure) to be broken by indirect force through bending that one of its ends must be fixed and the other end movable. The upper end of the fibula is fixed by its attachments at the superior tibiofibular joint and by the interosseous membrane; and to permit fracture by bending of its shaft, under such circumstances, by means of indirect force applied to the external malleolus, it is first of all necessary that the lower end of the fibula be freed from its attachments to the tibia. If these attachments are not freed, it is extremely unlikely that a fracture by bending can occur; though it is possible to conceive of an exceptional instance in which such an event might occur, as, for instance, if the surgical neck of the fibula (as in the second mechanism described by Bonnet) were pressed against the edge of a table or similar object by abduction of the foot; but in such a case the action of direct violence in causing the fracture could not be excluded. In life it would be less unlikely for a fracture by compression to occur (the force being transmitted in the long axis of the fibula which was still rigidly attached to the unbroken tibial shaft); whereupon the fracture would present a very different appearance (Fig. 15); or even less unlikely for a fracture by torsion to occur, though for this mechanism also it is necessary for the two ends of a bone to be movable in opposite directions or for one to be fixed and the other movable. Torsion was held by Maisonneuve (and no doubt correctly) to be the mechanism by which was produced the fracture in the upper third of the fibula described by him as "fracture par diastasis"; and it is this mechanism (torsion) which causes

it to be situated so close to the fixed end of the bone,[54] and which makes me class it as a variant of the fracture "by outward rotation of the foot" already discussed; and which convinces me that it does not belong in the same class with fractures produced by straight abduction of the foot, in which cases, I repeat, the fracture of the fibula occurs in its lower third and is caused by a bending mechanism.

FIG. 27.—Diastasis of inferior tibiofibular joint, following rupture of internal lateral ligament, but not succeeded by fracture of fibula (B, II, b, 1, Variant).

It is true, of course, that the force may cease to act before the fibula breaks; in which case merely a diastasis results (Fig. 27).

Careful study of thirty cases of fracture of the fibula through its surgical neck in our series has shown the characteristics of

[54] Once the lower end of the fibula is freed from the tibia, the interosseous membrane presents no obstacle to torsion.

a fracture by bending in all except two cases, in which it was clearly by torsion. When this lesion (by whatever mechanism) was unaccompanied by a diastasis of the inferior tibiofibular joint (or its equivalent, detachment of the anterior tubercle of the tibia), then the history has shown (in all but one case in

FIG. 28.—Fracture of lower fifth of fibula (surgical neck) by direct violence (kick of horse): there is no diastasis and under such circumstances the typical bending fracture (wedge detached from surface of flexion), which has been overcorrected by adduction of foot, could not have been produced by indirect violence (pressure of astragalus outward on external malleolus).

which the history is unknown) either that the fracture was due to direct violence (Fig. 28) or that the clinical signs of a tibiofibular sprain were present though no diastasis was shown by the roentgen ray. Quénu[35] (1912), however, for whose opinion

[35] Rev. de chir., 1912, xlv, 242.

every student of fractures has great respect, held that diastasis was not a necessary accompaniment of this type of fracture, though he acknowledged its extreme frequency, a fact to which he had called attention in 1909; and as early as 1907, he had pointed out that this type of fracture was that which was most frequently accompanied by diastasis. But he was at that time (1907) inclined to the view that the fracture of the fibula occurred simultaneously with or even before the diastasis. But Destot[56] (1912) is firm in his belief that fracture of the fibula above the inferior tibiofibular joint demands as a preliminary a sprain or a diastasis of that joint. Is not then the legend to Fig. 67, p. 142, in his monograph (1911) inaccurate when it states that the diastasis shown was possible only because of the existence of a fracture near the head of the fibula?

Finally, as in fractures by outward rotation, the most advanced stage of abduction fractures may be regarded as one in which the entire lower end of the tibia is torn off as the representative of the internal malleolus; but in these also the fibula breaks characteristically by flexion through its surgical neck (Fig. 29)—it is not a mixed oblique fracture as in the third degree of fracture by external rotation already described (Figs. 18 and 19).

Diastasis and Lateral Displacements of the Foot. It is well to analyze more carefully what is meant by diastasis of the inferior tibiofibular joint.[57] It is easily seen in the prepared specimen that in all movements of the foot there is greater strain on the anterior than on the posterior inferior tibiofibular ligament; and that if the anterior ligament alone is divided, a separation of the fibula from the tibia to the extent of about 1 cm. becomes possible anteriorly, the fibula still being attached by the interosseous ligament and the posterior tibiofibular ligament. This degree of separation is sufficient to constitute a diastasis; lesser degrees, with incomplete rupture of the anterior tibiofibular ligament, and therefore without separation, constitute a sprain.

[56] Lyon chir., 1912, viii, 245.

[57] The criteria of diastasis from a roentgenographic study will be discussed by Dr. Bromer in his appendix to this paper.

But in many cases in which a temporary diastasis (disjunction) may have been present at the moment of the accident, it is no longer present when the patient comes under the surgeon's care or is sent for roentgenologic study; and at the latter time can only be presumed to have existed by certain signs, especially evidences of a sprain fracture or detachment of the anterior tubercle of the tibia (Fig. 30).

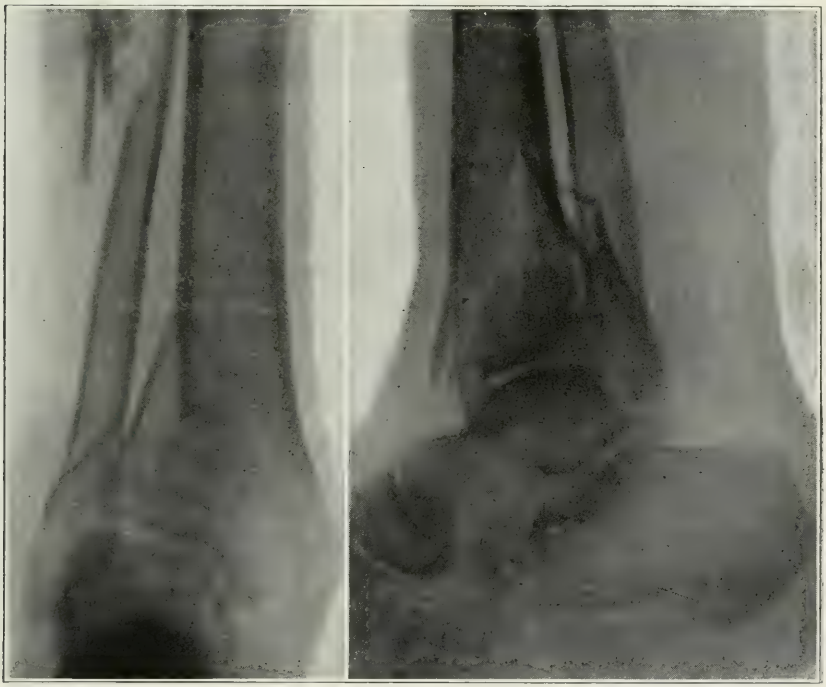


FIG. 29.—Fracture by abduction, third degree (B, III). The internal malleolus is represented by the whole lower end of the tibia; the fibula is broken by bending; there may or may not be an intermediate fragment. There were only two cases of this kind in our series of 300 cases.

If, in addition to division of the anterior tibiofibular ligament, one divides also the feltlike interosseous ligament, then a separation of the fibula from the tibia almost to the distance of 3 cm. may be possible; but even with this amount of diastasis of the anterior border of the fibula from the tibia, these bones are still

united by the posterior tibiofibular ligament, so that the fibula is not entirely freed from the tibia, though its lower end has become so movable as easily to permit a fracture by flexion.

FIG. 30.—Fracture by abduction (B, II, b, 1): Rupture of internal lateral ligament, followed by diastasis (note the intermediate fragment), and this succeeded by a bending fracture of fibula, in this case at an unusually high level.

Section of the posterior inferior tibiofibular ligament, as, indeed, noted by Quénu[58] (1907) permits only an insignificant separation of the fibula and tibia; and Quénu thinks that in life rupture of this ligament never occurs—at any rate it seems certain that this ligament is never ruptured alone, but only in conjunction with rupture of the anterior and interosseous ligaments. It is to be noted furthermore that it is next to an unheard of thing for the astragalus to be separated from the external malleolus, owing to the almost indestructible posterior band of the external lateral ligament; so that diastasis is not produced by a wedgelike action of the astragalus described by so many writers. I know of no case of true ascent of the astragalus between the tibia and fibula unaccompanied by the external malleolus: even in the beautiful case of diastasis recorded by Millikin,[59] though it is truly said that "the astragalus was jammed up between the outer surface of the tibia and the unfractured fibula," yet the astragalus had not been detached from the fibula, and therefore cannot have acted as a wedge in driving the bones apart, though it certainly acted as a prop to keep them asunder. I have been at pains to reproduce this lesion on the cadaver, demonstrating that it is quite possible for the astragalus to become lodged against the outer surface of the tibia, between the latter bone and the fibula, without rupture of the posterior band of the external lateral ligament; though, of course, it is necessary to divide the internal lateral ligament and the inferior (anterior and posterior) tibiofibular ligaments, as well as the interosseous ligament and the interosseous membrane (the latter as far up as the upper third of the leg). And unless, in addition to the above, the anterior and middle bands of the external lateral ligament were divided, the astragalus maintained itself only in very unstable equilibrium between tibia and fibula; though no doubt in life, the stability would be greater owing to muscular tension. Moreover, in the type of fracture with diastasis in which the fibular fragment is accompanied by a fragment detached from the external surface

[58] Rev. de chir., 1907, xxxv, 897.
[59] Ann. Surg., 1919, lxix, 650.

of the tibia (as in Cooper's illustration copied by Vidal de Cassis), and which is usually described as exhibiting ascent of the astragalus between the bones of the leg, or along the outer side of the tibia, the astragalus carries with it the external malleolus (the lower fibular fragment), so that in no true sense has the astragalus ascended between the bones. Of course, it is true that dislocation of the astragalus may occur, forward or backward, detaching it from the fibula; and there is no denying the possibility of its being dislocated upward between the intact tibia and fibula. All that I contend is that for it to become detached from the external malleolus in fractures of the leg bones at the ankle must be extremely rare, as there does not appear to be any such case on record.[60]

The Posterior Marginal Fragment of the Tibia, and Posterior Displacement of the Foot. The existence of this fragment as the sole osseous lesion in a certain number of cases now on record,[61] including one of our own, proves that it may be the earliest stage of a lesion involving the ankle-joint. But as there never has been any displacement of the fragment in these isolated lesions, it cannot be considered of much importance unless associated with a fracture of the fibula. As already noted, the existence of this fragment as a complicating lesion was well known to Cooper; it was noted by Earle[62] (1828) and by Adams[63] (1835); it was observed at autopsy by Dupuytren "with surprise," according to Malgaigne[64] (1832); Thaon[65] presented an autopsy specimen and said he had often seen this fragment in experiments made by Tillaux; it was clearly and accurately if succinctly described by Nélaton,[66] (1874), and the

[60] Gross (System of Surgery, ed. 5, Philadelphia, 1872, ii, 85), says Druitt refers to such a case, but I have been unable to find the original report to ascertain the exact lesions. Wendel (Beitr. z. klin. Chir., 1898, xxi, 146) collected five cases of upward dislocation without fracture; but the exact lesions do not appear to have been determined definitely in any case.

[61] Tanton (footnote 29, p. 660) refers to twenty cases recorded prior to 1916. As an isolated lesion it was first observed by Meissner (Beitr. z. klin. Chir., 1909, lxi, 136).

[62] Lancet, 1828-1829, ii, 346.

[63] Adams in Todd: Cyclopedia of Anatomy and Physiology, London, 1835-1836, i, 161.

[64] Gaz. méd. de Paris, 1832, iii, 647.

[65] Bull. Soc. anat. de Paris, 1870, xlv, 212.

[66] Eléments de path. chir., ed. 2, Paris, 1874, iii, 296.

fragment was recognized as a serious complication by Edmund Andrews[67] (1883, 1897). Since the introduction of the roentgen ray, it has been studied by Chaput[68] (1899, 1907), Bondet[27] (1899), Grashey[69] (1907), Meissner[61] (1907), Plagemann[70] (1911), Destot[8] (1911), Quénu (1912-1915), and by Stimson (in every edition of his book since 1899). Hence it was with surprise that surgeons who were tolerably familiar with the literature of their profession, as well as with fractures of the ankle, saw Cotton[71] (1915) describe it as "a new type of ankle fracture" which "has never been adequately described in print and has apparently escaped the notice even of those who deal with fractures habitually"; and noticed his complacent comment that in certain circles it was called "Cotton's fracture"; as well as his statement that he believed there were no autopsy specimens.[72] Cotton, however, did well to call attention to its frequency; as did Speed in a paper which was not published until after Cotton's paper was read, though it appeared in print before the latter.

The mechanism by which this fracture is produced is almost certainly, as contended by Lucas-Championniére,[73] a crushing force from below upward;[74] it is possible that traction by the posterior inferior tibiofibular ligament, through the medium of the fractured lower end of the fibula, may aid in displacing the fragment, even if it cannot be the sole cause of its detachment.

[67] Andrews in Ashhurst: International Encyclopedia of Surgery, New York, 1883, iii, 707, also in Internat. Clinics, Philadelphia, 1897.

[68] Bull. et mém. Soc. de chir. de Paris, 1899, xxv, 776; Les fractures malleolaires, Paris, 1907.

[69] Fortschr. a. d. Geb. d. Röntgenstrahlen, 1907, xi, 152.

[70] Beitr. z. klin. Chir., 1911, lxxiii, 688.

[71] A New Type of Ankle Fracture, Jour. Am. Med. Assn., January 23, 1915, lxiv, 318.

[72] In addition to the autopsy specimen which figures in Cooper's Plate XVII, (reproduced here as Fig. 3), those described by Stimson and that of Thaon, already alluded to, there are in the Mütter Museum of the College of Physicians of Philadelphia three autopsy specimens showing this posterior marginal fragment. The most recent of these specimens has been in the museum for a period at least of forty years. (A description of these specimens will be published elsewhere by Dr. Bromer and myself.)

[73] Bull. Soc. anat. de Paris, 1870, xlv, 212.

[74] Rochet (Rev. d'orthop., 1890, i, 269) produced it experimentally by dropping a weight of 60 kilograms, from a height, on the upper end of the tibia while the foot was in plantar flexion.

The size of the fragment varies from a small portion of the lip to a large fragment, extending 10 cm. up the posterior surface of the shaft. McKnight, at the meeting of the Philadelphia Academy of Surgery, May 2, 1921, showed a roentgenogram (anteroposterior view only) of a fracture which I believe conformed to this type, though the fragment was the largest with which I am acquainted: the fragment (an isolated lesion, without displacement) included nearly all the posterior lip of the tibia, as well as its entire lateral (fibular) border; and the apex of the large wedge extended to a point about 10 cm. above the articular surface, on the posterolateral border of the tibia.

TABLE I

INCIDENCE OF POSTERIOR MARGINAL FRACTURES

	Total ankle fractures.	Posterior marginal fractures.			
		Associated.	Isolated.	Total.	Per cent.
Ashhurst and Bromer	300	57	1	58	19
Chaput (Les fractures malléolaires, Paris, 1907)	136	42	0	42	30
Destot (Quénu: Bull. et mém. Soc. de chir. de Paris, 1913, xxxix, 165)	1700	139	6	145	8.5
Quénu (Rev. de chir., 1912, xlv, 260)	129	11	1	12	9.3
Sear (Med. Jour. Australia, 1917, i, 526)	156	26	3	29	18.6
Speed (Surg., Gynec. and Obst. 1914, xix, 73	161	16	0	16	10.0

INCIDENCE OF ASSOCIATED POSTERIOR MARGINAL FRACTURES

Type.	Ashhurst and Bromer.		Destot.	
	No.	Per cent.	No.	Per cent.
Fibula, oblique mixed	0	..	0	
Low Dupuytren	51	88	89	64
Bimalleolar	0	..	17	12
Pott's	5	8.6	29	20
Adduction	1	1.7	0	
Maisonneuve	0	..	4	3
	57		139	

The lesion corresponds, as Tanton[29] (*loc. cit.*, p. 171) has noted, to Rhea Barton's fracture of the posterior margin of the radius.

It is a much more frequent complication than commonly supposed. Among our 300 cases it was present no less than fifty-eight times, or in 19 per cent of all the cases; and in 51 or 50 per cent, of those conforming to the "low Dupuytren" type.

There is sometimes confusion between this posterior marginal fragment and the intermediate fragment ("third fragment of Tillaux"), as remarked before.[23] The posterior tubercle of the tibia, which limits posteriorly the gutter for the reception of the fibula (Fig. 4), and to which is attached the posterior tibiofibular ligament, may be fractured by the pull of this ligament. It appears then in lateral roentgenograms as an infraction or sprain fracture, but does not involve the ankle-joint. In the true marginal fractures, on the other hand, the line of fracture always extends into the tibial plafond; the fragment (contrary to what is said by Tanton) usually remains attached to the external malleolus by the posterior tibiofibular ligament, and is often displaced backward with the external malleolus; sometimes (more frequently than thought) it may be detected in anteroposterior roentgenograms as a deltoid fragment; and, if large, its shadow sometimes overlaps that of the median border of the tibia, giving a double contour.

The posterior marginal fragment, as already remarked, may occur as an isolated lesion; only once in our series (Fig. 44). It may be associated with (*a*) merely a diastasis, with sprain fracture of the anterior tubercle of the tibia (Fig. 31); (*b*) it may accompany the mixed oblique fracture of the fibula in the various stages of the fracture by outward rotation, being small (Fig. 32), medium sized (Fig. 33), or large (Fig. 34); and (*c*) it occurs also with fractures by abduction (Figs. 35 and 36), though very much less frequently than with external rotation fractures. Quénu has had one case in which fracture of the internal malleolus was the only other lesion, and another fracture

of precisely the same type has come under our notice since our series of 300 cases was completed.

Its presence is not necessary to permit the occurrence of posterior displacement of the foot (Figs. 16 and 25), but certainly

FIG. 31.—Fracture of posterior margin of tibia, associated with a tibiofibular diastasis, as evidenced by sprain fracture at this joint. The posterior marginal fragment, which is unusually large, is visible also in the anteroposterior view.

favors it. This posterior displacement is very rare without the posterior marginal fragment. Either a posterior marginal fragment must exist, or there must be rupture of the posterior inferior tibiofibular ligament. It is only a continuance of the force (now the weight of the body) after the fracture has been

produced, that causes the displacement, since there are in our series no less than fourteen instances (out of a total of fifty-eight posterior marginal fractures) with slight if any displacement.

The factors which permit posterior displacement of the foot deserve a few words. Quénu was the first, I believe, to point

FIG. 32.—Small posterior marginal fragment in a case of fracture by external rotation. Notice the mixed oblique fracture of fibula; the intact internal malleolus (rupture of internal lateral ligament), and the complete posterior dislocation of the foot, the astragalus and the posterior marginal fragment accompanying the external malleolus.

out that the essential lesion is freeing the lower end of the fibula from the tibia; and I may remark that this necessity is merely a corollary of what has been insisted on so often in these pages, namely, the indissolubility of the union between the astragalus and the fibula. I cut everything else at the ankle (tendons and ligaments as well as all other soft parts) leaving only the

middle and posterior bands of the external lateral ligament attaching the external malleolus to the foot. Under these circumstances even an incomplete posterior dislocation of the foot cannot occur (except by rotation of the foot inward around the long axis of the leg, a mechanism which does not occur in life). I then cut the middle band of the external lateral ligament;

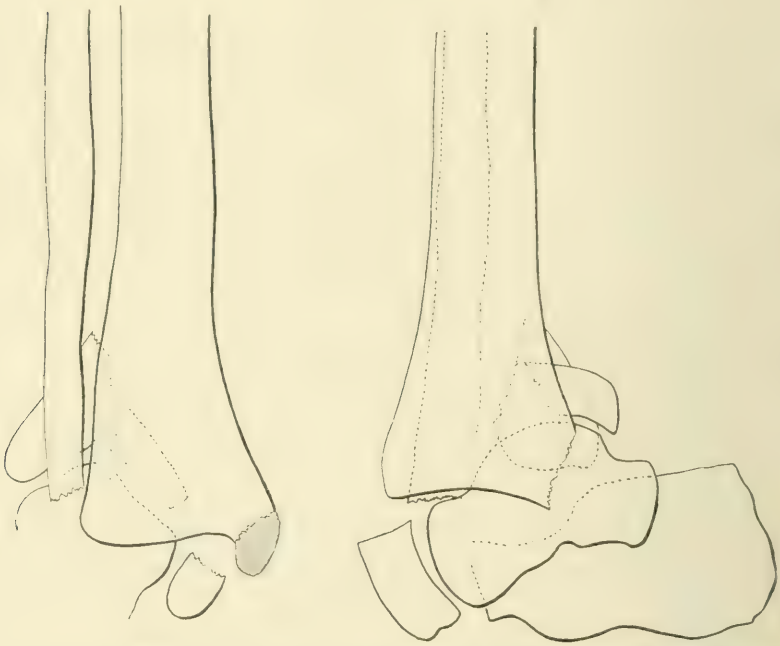


FIG. 33.—Medium-sized posterior marginal fragment in association with mixed oblique fracture of fibula and fracture of internal malleolus. The posterior marginal fragment, which is visible also in the anteroposterior view, accompanies the external malleolus and the astragalus in the very marked lateral displacement and in the incomplete posterior dislocation.

but still no posterior displacement could be produced. Next I fractured the fibula 7.5 cm. (3 inches) above its tip; but this did not permit posterior displacement. Finally, I divided the anterior inferior tibiofibular ligament, and even the interosseous ligament; but so long as the fibula remained attached to the tibia, and the astragalus to the fibula, no posterior dislocation

could be produced. On another foot, I divided all structures uniting the foot to the fibula except the middle band of the external lateral ligament; but no dislocation of the foot was

FIG. 34.—Large posterior marginal fragment in association with mixed oblique fracture of fibula and fracture of internal malleolus; only moderate displacement, chiefly due to the outward rotation.

possible; then I divided also the posterior inferior tibiofibular ligament and the interosseous ligament (which allowed less diastasis than when the anterior tibiofibular ligament was divided), but still no posterior dislocation of the foot was pos-

sible except by rotating it inward around the one remaining ligament as a pivot.

As a result of these investigations, it may be concluded that either the middle or the posterior band of the external lateral ligament is sufficient to hold the astragalus against the external

FIG. 35.—Small posterior marginal fragment in association with abduction fracture of fibula and fracture of internal malleolus. Note the diastasis, the typical wedge detached from the flexion surface of fibula and the posterior marginal fragment accompanying the external malleolus and astragalus in their marked lateral and posterior displacement.

malleolus, and that (even after fracture of the internal malleolus or rupture of the internal lateral ligament) *no backward dislocation of the foot can occur* (1) unless the astragalus is freed from the external malleolus by rupture of both the middle and posterior bands of the external lateral ligament (a lesion which

apparently has not been recorded in association with fractures at the ankle); (2) or unless the external malleolus is freed from the tibia (*a*) by diastasis of the inferior tibiofibular joint, with

FIG. 36.—Posterior marginal fragment associated with diastasis and fracture of internal malleolus, and with fracture of fibula by bending backward (flexion surface posterior, extension surface anterior), as evidenced by wedge detached from posterior surface of fibula.

or without detachment from the tibia of an intermediate fragment; (*b*) by fracture of the fibula in such a way as to detach with the lower fragment of the fibula the fibular insertions of both the middle and posterior bands of the external lateral

ligament (in other words, unless a virtual diastasis occurs, but one in which the intermediate fragment belongs to the fibula instead of to the tibia). One very frequent fracture of the

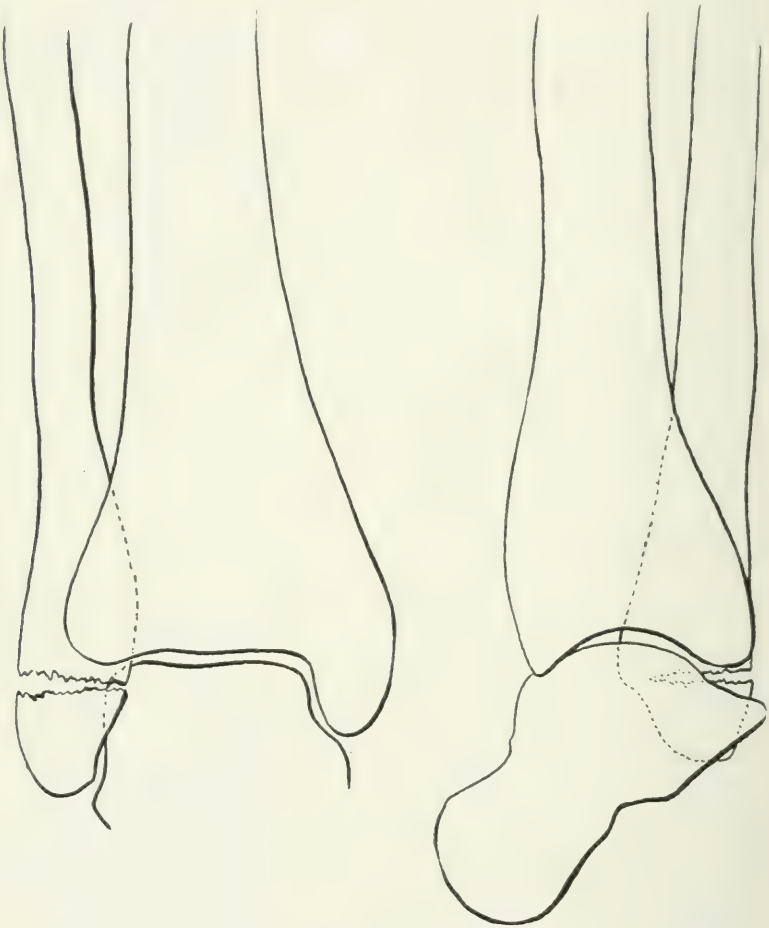


FIG. 37.—Fracture by adduction, first degree (C, I); avulsion of external malleolus. See page 715.

fibula (the mixed oblique of Destot) fulfils these requirements; as does another less usual, namely, a transverse fracture of the external malleolus proper below the level of the tibia. But

as the latter fracture usually is subperiosteal or incomplete, and without displacement, it follows that posterior displacement of the foot with this lesion is unknown.

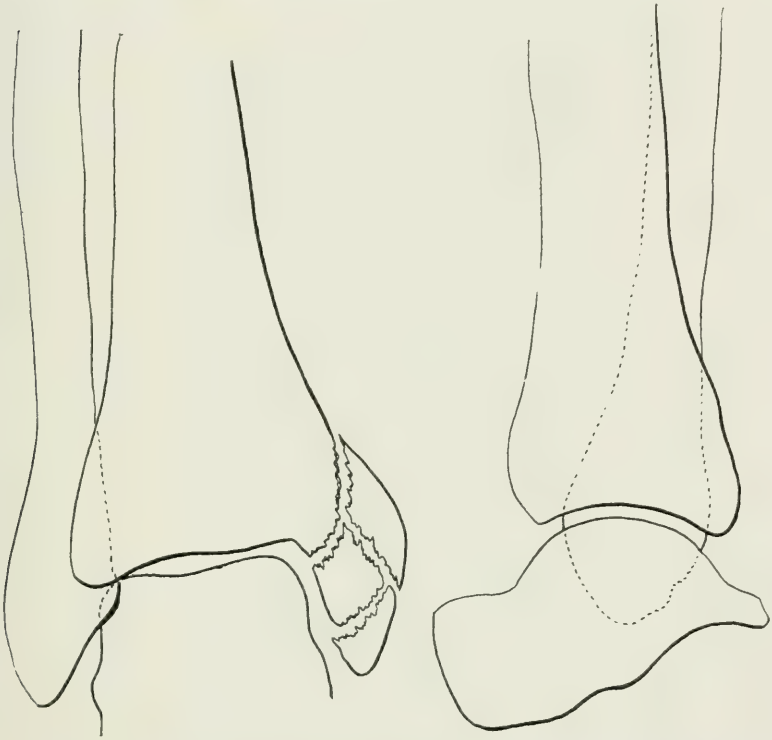


FIG. 38.—Adduction fracture, second degree (C, II, a); avulsion of external malleolus, represented by partial rupture of anterior fibers of external lateral ligament (rare) followed by compression fracture (note the comminution) of internal malleolus.

Therefore, the fractures which may be accompanied by posterior displacements of the foot are those of the Pott or Dupuytren type in which diastasis is the rule; and those of the low Dupuytren variety, in which the fracture of the fibula is of the mixed oblique type. Quénu[75] is wrong, I am sure, in

[75] Rev. de chir., 1912, xlvi.

claiming (1912) that the existence of a posterior marginal fragment is a necessary condition for the occurrence of a posterior subluxation (Figs. 16 and 25); but it is well to remember that

FIG. 39.—Adduction fracture, second degree (C, II, a); avulsion of external malleolus followed by compression fracture of internal malleolus.

the distorted shadows of a roentgenogran in a case of fracture of the low Dupuytren variety, in any of its stages, may mislead the observer into the belief that a posterior displacement

is present, when the appearances are due entirely to an outward rotation of the astragalus around the long axis of the leg.

Forced Movements of Adduction (Tibial Flexion). Since the time of Cooper and that of Maisonneuve and Bonnet, there has been little dispute about the mechanism of these fractures:

FIG. 40.—Adduction fracture, second degree (C, II, b); large tibial fragment extending into shaft replaces compression fracture of internal malleolus.

it has been generally recognized that a tearing off of the external malleolus is the first lesion (Fig. 37), followed by a compression fracture of the internal malleolus (Figs. 38 and 39); or, when the weight of the body forces the tibia heavily on the displaced astragalus, a splitting upward of the tibial shaft occurs, the

line of fracture commencing at some point of the articular surface, splitting this in the sagittal plane and terminating on the median border of the tibia at a variable distance above the

FIG. 41.—Adduction fracture, second degree (C, II, b); splitting fracture of median articular surface of tibia replaces crush of internal malleolus. See page 716.

internal malleolus (Figs. 40, 41 and 42). The more nearly longitudinal the line of fracture, the less necessary will it be for the external malleolus to be fractured as a preliminary step: such

fractures verge into those due to comminution upward in the long axis of the limb. Stimson[76] (1912) gives an admirable illustration of such a longitudinal fracture of the tibia without

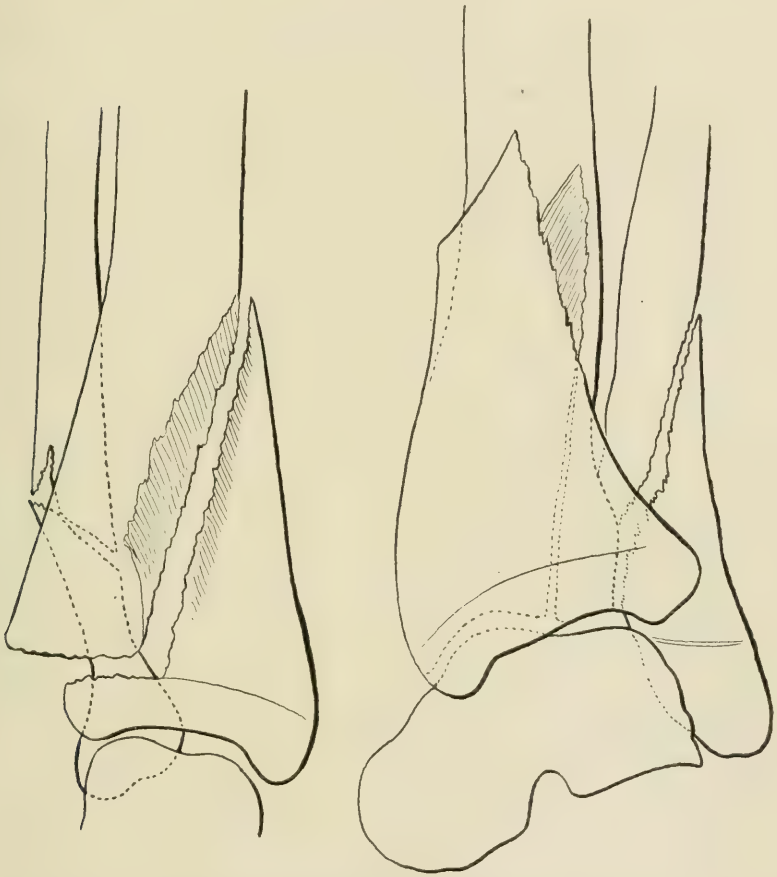


FIG. 42.—Adduction fracture, third degree (C, III); detachment of lower epiphysis with splitting of median surface of tibia.

any lesion of the fibula. But if the displacement in these longitudinal fractures is marked enough, the fibula may break secondarily (by flexion or torsion) above the inferior tibiofibular joint,

[76] Fractures and Dislocations, ed. 7, Philadelphia, 1912, Plate 27.

as the lower end of the fibula is carried inward with the astragalus (Fig. 41). A variant of this type is the lesion recorded by Silhol,[77] in which the fibular fracture is replaced by an inter-

FIG. 43.—Adduction fracture, third degree (C, III); the supramalleolar fracture by adduction (type produced experimentally by Tillaux). See pages 657 and 719.

mediate fragment detached from the tibia. Even in one of our own cases (Fig. 40), there is an intermediate fragment, in addition to the fracture of the fibula.

[77] Bull. et mém. Soc. de chir. de Paris, 1916, xlii, 819.

The supramalleolar fracture by adduction, produced experimentally by Tillaux, and already mentioned at p. 657, may be considered the most advanced degree of this type (Figs. 42 and 43).

Summary

These then are the abnormal movements—external rotation, abduction, adduction—which are responsible for the great majority (95 per cent) of fractures about the ankle: external rotation causes about 61 per cent, abduction about 21 per cent, and adduction about 13 per cent of the lesions. The remaining small proportion (5 per cent) consists chiefly of those fractures which may be recognized either as caused by compression in the long axis of the limb, or by direct crushes; or even by very rare forced movements such as straight flexion or extension, internal rotation, etc. The fractures by compression in the long axis of the limb include (*a*) the isolated fractures of the tibial margins (anterior, posterior (Fig. 44), median or even lateral), which Sear[78] speaks of as "vertical plane fractures"; (*b*) comminuted fractures of the tibial plafond (Fig. 45), and (*c*) T or Y-fractures involving the ankle-joint (Figs. 46 and 47), which may be regarded as an advanced degree of the supramalleolar V-fractures described by Gosselin,[79] the latter usually being complicated by a fissure extending into the ankle-joint, the mechanism being the same as in those of the radius with comminution of the wrist fragment.[80] The supramalleolar fractures of Malgaigne[81] (1847) are thus distributed according to their mechanism, some into those by adduction, others as due to compression in the long axis of the limb, and a few probably due to direct violence (Fig. 48). We count no supramalleolar fracture as one involving the ankle unless it falls within 4 cm. of the joint level (Richet, 1875). Fractures as

[78] Med. Jour. Australia, 1917, i, 526.
[79] Gaz. d. hôp., 1855, xxviii, 218.
[80] Gosselin: Bull. et mém. Soc. de chir. de Paris, 1863, v, 147.
[81] Fractures, Paris, 1847, p. 818.

close to the ankle joint as 4 cm. compromise its functions as surely as do supracondylar fractures of the humerus compromise those of the elbow.

Now, it is because of the impossibility of classifying together anatomically lesions which, as Stimson says, are merely alter-

FIG. 44.—Isolated fracture of posterior tibial margin; no displacement; from compression upward and backward.

native, or whose differences are due to the early cessation of the force before the typical form has been reached; or, I may add, to its continuance after the typical stage has been passed—it is because of this impossibility, I repeat, that I believe a classification based on mechanism, imperfect though it be, is, nevertheless, more easily understood and remembered. And it is

for this purpose that Dr. Bromer and I have ventured to arrange our series of fractures as shown in Table II (p. 725). Our aim has been to place under each mechanism, first, the simplest resulting

FIG. 45.—Comminuted fracture of tibial plafond; from compression upward.

form of fracture, and to advance thence to more complicated lesions, noting at the same time in their appropriate places the variants and the complications of the simple or the more complex lesions which were encountered. It is true that it is not

always easy to determine the mechanism, even with all the aid derived from the clinical history, the roentgen ray and a knowledge of the lesions which can be produced on the cadaver; but the more one studies the subject, the fewer exceptions he will find to the general laws of mechanics; and the more his experience increases, the easier will it become to recognize the

FIG. 46.—T or Y-fracture involving ankle joint; from compression upward.

variants from the typical lesions. Those fractures which have given us most concern are the true bimalleolar fractures with little or no displacement, since these may be caused possibly by external rotation, certainly both by abduction and adduction. But as the total number of these fractures observed is small (sixteen cases or only 5 per cent of the entire series), and as even among this number the mechanism was reasonably certain

in all but ten cases (3 per cent of the entire series), the margin of error is small.

It will be noted that in our classification the fibular lesion dominates the clinical picture in the first two classes (those of outward rotation and abduction), and that these correspond to the first grand division of Destot's classification—those fractures

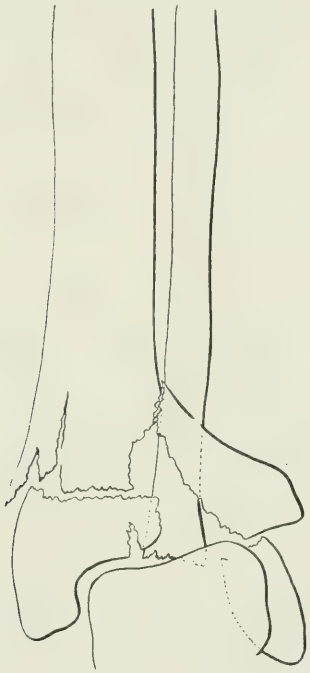


FIG. 47.—T or Y-fracture involving ankle-joint; from compression upward.

in which the equilibrium of the foot is involved; while the third, fourth and fifth classes correspond to Destot's second division—those fractures which involve the tibial pestle and compromise the function of support. For the sake of completeness we also give an anatomopathologic classification, constructed on the same principles (Table III).

Following the classifications is a catalog of the lesions encountered in this series of cases, which, with the aid of the classi-

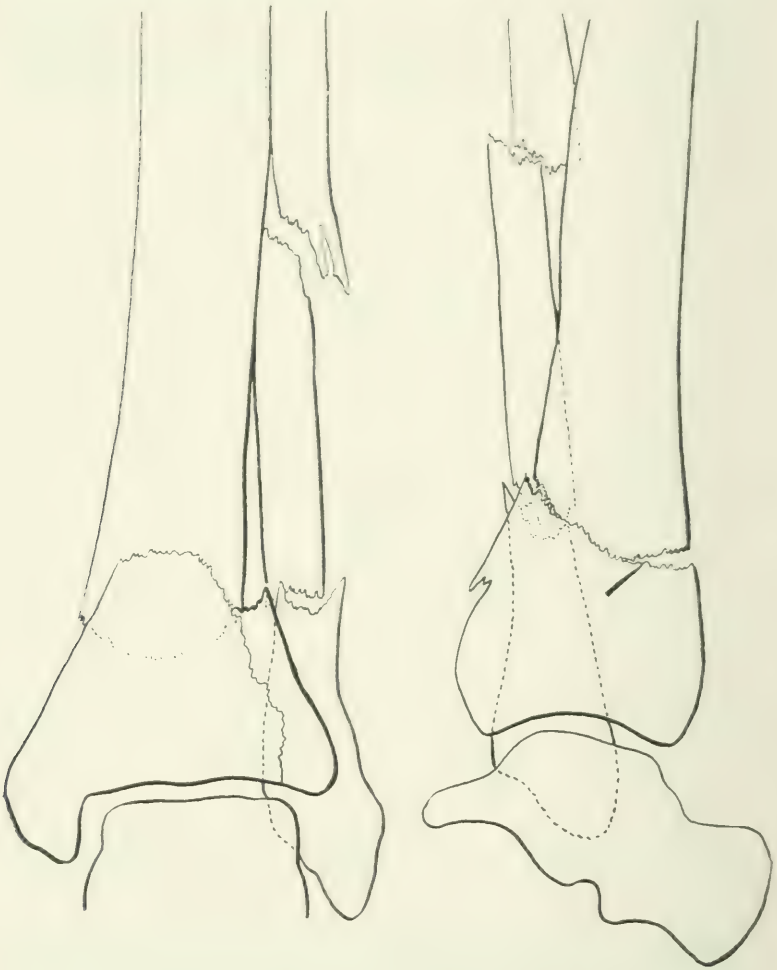


FIG. 48.—Comminuted supramalleolar fracture; probably by direct violence.

fications as a guide, we hope may prove of interest to students of fractures (Table IV).

TABLE II

CLASSIFICATION OF THREE HUNDRED ANKLE FRACTURES

	Cases.	Per cent.
A. *Fractures by External Rotation.*		
1. First degree: Lower end of fibula only ("mixed oblique")	79	26.00
2. Second degree: Same, *plus* rupture of internal lateral ligament or fracture of internal malleolus ("low Dupuytren"), viz.	100	33.00
(*a*) Internal lateral ligament, uncomplicated . . 13		
Internal lateral ligament complicated by posterior marginal fragment of tibia . . 13		
(*b*) Internal malleolus, uncomplicated . 32		
Internal malleolus complicated by posterior marginal fragment of tibia . . 42		
3. Third degree: Same, *plus* fracture of whole lower end of tibia, representing the internal malleolus	5	1.70
Total fractures by external rotation	184	61.00
B. *Fractures by Abduction* (*Fibular Flexion*).		
1. First degree: Internal malleolus only	19	6.60
2. Second degree: Same, *plus* fracture of fibula (transverse, above or below tibiofibular joint)	42	13.70
(*a*) Below inferior tibiofibular joint (no diastasis) ("bimalleolar fracture") . 14		
(*b*) Above the inferior tibiofibular joint (with diastasis) ("Pott's fracture," "Dupuytren type") . . 28		
3. Third degree: Internal malleolus represented by whole lower end of tibia	2	0.66
Total fractures by abduction	63	21.00
C. *Fractures by Adduction* (*Tibial Flexion*).		
1. First degree: External malleolus only, transverse, at or below level of tibial plafond	27	9.00
2. Second degree: Same, *plus*		
(*a*) Internal malleolus below level of tibial plafond ("bimalleolar fracture") . 3		
(*b*) Median surface of tibia up and in from joint surface . . 8	11	3.60
3. Third degree: Same, *plus* whole lower end of tibia ("supramalleolar fracture by adduction")	2	0.66
Total fractures by adduction	40	13.30
D. *Fractures by Compression in Long Axis of Leg.*		
1. Isolated marginal fractures . . 1		
2. Comminution of tibial plafond . . 3		
3. T or Y-fractures ("V-fractures of Gosselin") 4		
Total fractures by compression in long axis of leg	8	2.7
E. *Fractures by Direct Violence* (*Supramalleolar types*).	5	1.7

TABLE III

ANATOMOPATHOLOGIC CLASSIFICATION OF THREE HUNDRED ANKLE FRACTURES

	Cases.	Per cent.
A. *Fibula below Inferior Tibiofibular Joint.*		
1. Alone (slight or no displacement, often subperiosteal)	27	9.0
2. Same, *plus* internal malleolus below tibial plafond (includes bimalleolar fractures by adduction and by abduction)	16	5.0
3. Same, *plus* split of tibia up and in	8	2.6
	51	17.0
B. *Fibula, Obliquely through Inferior Tibiofibular Joint.*		
1. Alone (slight or no displacement, often subperiosteal)	79	26.0
2. Same, *plus* rupture of internal lateral ligament	26	9.0
3. Same, *plus* fracture of internal malleolus	74	25.0
	179	60.0
C. *Fibula, above Inferior Tibiofibular Joint.*		
1. Alone (slight or no displacement, often subperiosteal)	0	
2. Same, *plus* rupture of internal lateral ligament and diastasis	8	2.7
3. Same, *plus* fracture of internal malleolus and diastasis	20	6.6
	28	9.3
D. *Tibia, Involving Ankle-joint.*		
1. Internal malleolus alone (slight or no displacement, often subperiosteal)	20	6.6
2. Isolated marginal fractures	1	.33
3. Comminuted, T and Y-fractures	7	2.3
	28	9.3
E. *Supramalleolar Fractures, Not Involving Ankle-joint Directly*	14	4.3

TABLE IV

CATALOGUE OF THREE HUNDRED FRACTURES INVOLVING THE ANKLE

	No. Cases.
A. *By Rotation of the Astragalus Outward Around the Long Axis of the Leg.*	
1. First degree: Lower end of fibula only. Oblique or spiral fracture from above and behind, down and front, the line of fracture passing through the inferior tibiofibular joint. Line varies from nearly transverse to nearly longitudinal, but is always higher on posterior than on anterior surface of fibula. In 84 per cent of cases the lower anterior end of line of fracture was on anterior surface of external malleolus between its tip and the anterior inferior tubercle of the tibia; in 8 per cent of cases it was above the anterior tubercle (when there was diastasis or the tubercle was detached); and in 8 per cent it passed through the fibula at the level of the anterior tubercle on the tibia. Slight or no displacement . . .	76
Variant: Fracture of fibula in *upper third* from lesion at ankle (Maisonneuve, 1840) . . . . . (In this case there was incomplete fracture of fibula below its head (Fig. 17) but without bony lesion at ankle.)	1
Complication: Intermediate fragment	2
	— 79
2. Second degree: Fibula as in first degree, *plus* rupture of internal lateral ligament or fracture of internal malleolus. There may be slight or considerable lateral displacement, but in uncomplicated cases there is rarely a real posterior displacement, the apparent posterior displacement seen in lateral roentgenographic views being due usually to outward rotation, which distorts the shadows.	
(*a*) Internal lateral ligament (in roentgenograms this lesion is differentiated from first degree only by presence of lateral displacement of astragalus from internal malleolus with slight displacement of lower fibular fragment)	12
Variant: Diastasis without fracture of fibula (Fig. 16)	1
First complication: Fracture of posterior tibial margin. Line of fracture usually extends about 2 cm. up along posterior surface of tibia, and running down thence nearly vertically, detaches a mere chip or more often one-fourth to one-third of the articular surface; rarely as much as one-half	10
Variant: Diastasis without fracture of fibula, but with posterior marginal fragment (Fig. 31)	1
Second complication: Intermediate fragment . . .	0
Third complication: Second fracture of fibula in upper third (Maisonneuve's fracture; these two cases occurred in opposite legs of same patient) . . .	2
(*b*) Internal malleolus . . . . . (With slight or no displacement, 20 cases)	32
First complication: Fracture of posterior tibial margin (Figs. 32, 33, 34) . . . . . (Slight or no displacement, 14 cases) (Lateral and posterior displacement, 26 cases)	40
Second complication: Intermediate fragment . . .	2
	— 100

TABLE IV—(*Continued*)

	No.	Cases.
3. Third degree: Internal malleolus represented by whole lower end of tibia . . . . . (This will include most cases of separation of lower epiphysis of tibia.)		5
Total fractures by mechanism of outward rotation . .		184
B. *By Abduction (Fibular Flexion) of Foot.*		
1. First degree: Fracture of internal malleolus only, transverse, at or below level of tibial plafond. No displacement		19
2. Second degree: Rupture of internal lateral ligament or fracture of internal malleolus, as in first degree, followed by fracture of fibula more or less transverse either below or above inferior tibiofibular joint.		
(*a*) Fracture of fibula below inferior tibiofibular joint (*i. e.*, of external malleolus proper) with fracture of internal malleolus or rupture of internal lateral ligament	13	
(With no displacement, 3 cases; internal malleolus displaced more than external, 4 cases; both malleoli equally displaced, 6 cases)		
Variant: Crush of calcaneum (replacing fracture of internal malleolus) followed by compression fracture of external malleolus	1	
(*b*) Fracture of fibula above inferior tibiofibular joint (in lower third), the line of fracture indicating a fracture by flexion (Biegungsbruch), with slight or no torsion; slightly oblique, often comminuted with detachment of a wedge-shaped fragment from the surface of flexion and its apex toward the surface of extension	28	
	—	42
Viz.:		
1. Fracture of fibula alone, or with rupture of internal lateral ligament	6	
Variant: Diastasis without fracture of fibula	2	
2. Fracture of fibula with fracture of internal malleolus at or below level of tibial plafond	12	
First complication: Posterior marginal fragment of tibia (Figs. 35 and 36) . . .	5	
Second complication: Intermediate fragment	3	
3. Third degree: Internal malleolus represented by whole lower end of tibia (this will include some cases of separation of lower epiphysis of tibia)		2
Total fractures by mechanism of abduction		63

	No.	Cases.
C. *By Adduction (Tibial Flexion) of Foot.*		
1. First degree: Fracture of external malleolus, transverse, at or below level of tibial plafond. Slight or no displacement, often subperiosteal	26	
Complication: Posterior marginal fragment of tibia (infraction)	1	
	—	27
2. Second degree: Fracture of external malleolus as in first degree, *plus*:		
(*a*) Fracture of internal malleolus below level of tibial plafond	2	
Variant: Fracture of external malleolus represented by rupture of external lateral ligament	1	
(*b*) Fracture of median surface of tibia up from tibial plafond	8	
	—	11
3. Third degree: Fracture of fibula usually above inferior tibiofibular joint, *plus* fracture across tibia above ankle-joint (Tillaux, 1872)		2
Total fractures by mechanism of adduction		40
D. *By Compression in Long Axis of Leg.*		
1. Isolated marginal fractures of tibia: Anterior margin, none; posterior margin, 1; median margin, none; lateral margin, none	1	
2. Comminution of tibial plafond	3	
3. T or Y-fractures of tibia into ankle-joint	4	
	—	8
E. *Fractures by Direct Violence (Supramalleolar Types)*		5

Grand Total, 300

Roentgen-ray Study of the Ankle-joint

By Dr. Bromer.

I. The Normal Ankle-joint—Anatomy

By means of the roentgen ray, the structure of the ankle-joint can be most satisfactorily demonstrated. The tibiofibular mortise is shown (Fig. 49), and below it the trochlea of the astragalus fitting into it as a tenon. The "plafond" and the "cheeks" of the mortise are easily recognized, likewise the longer external malleolus and the anterior and posterior tubercles of the fibular groove. In the lateral view (Fig. 50), the low pro-

jection of the posterior lip, called by Destot the posterior malleolus, reinforcing the mortise posteriorly, can easily be visualized; and the point where it meets the trochlear surface of the astragalus acting as a check on the latter when the foot is in plantar flexion, as in walking, is most noticeable. (The degree of plantar flexion

FIG. 49.—Anteroposterior view of ankle-joint. See page 729.

in the roentgenogram is much greater than is assumed in walking, but is used more fully to demonstrate the check.) In this view, the longer external malleolus is shown overlapping the shadow of the internal malleolus. With stereoscopic plates they usually can be fairly well distinguished. The lateral tubercle on the posterior surface of the astragalus (os trigo-

num) is the farthest posterior shadow of the astragalus. In the interpretation of roentgenograms of the ankle-joint, due care must be exercised not to diagnose a sprain-fracture of the posterior tubercle, where an accessory bone, the os trigonum, often is found. In general, no fracture exists, and it is this bone that is present if the surfaces of the fragment are smooth and rounded.

FIG. 50.—Lateral view, plantar flexion.

II. FUNCTION AND MOVEMENT OF THE ANKLE-JOINT

The roentgen-ray can be used to demonstrate the movements of the ankle-joint. Thus the rotation of the astragalus about

the axis previously described by Dr. Ashhurst in full dorsiflexion and plantar flexion is shown in Figs. 50 and 51. The movements of the fibula were shown in the following way: A normal ankle was examined: the ankle and leg of the subject being securely bound to the table, a Bowen stereoscopic plate holder was placed beneath the ankle-joint. Lateral views were made first with the foot in full dorsiflexion, then later with the foot in full plantar flexion. Great care was exercised that

FIG. 51.—Lateral view, dorsiflexion.

the position of the leg *i. e.*, tibia and fibula—was not altered. The stereoscopic plate holder gave an exact duplicate position for the second exposure. The tube was in no way shifted. Anteroposterior views were taken in the same way. In this plane the heel was allowed to project as far as possible over the edge of the plate holder in order to obviate any change in height of the tibia above the plate due to change of position of the os calcis. The superior tibiofibular articulation was examined in the same way.

It was found that these views could be superimposed over diffused strong light. As an additional check the distances between the same relative points were accurately measured. The fact that the images of the tibia could be accurately superimposed would tend to rule out any possibility of error due to change in height of tibia. It was found that in full plantar flexion in the anteroposterior view the fibula moved inward and downward 1 mm. or, *vice versa*, so much expansion and movement upward occurred in full dorsiflexion. No change of the fibula in relation to the tibia could be found in the lateral views.

With regard to the upper extremity of the fibula, the forward movement of the fibula in plantar flexion was shown in the lateral view by an increase of 1 mm. in the distance between the posterior lip of the upper extremity of the fibula and the posterior border of the tibia. In the same way in the anteroposterior view the head of the fibula was found lying farther in toward the midline with the foot in plantar flexion than it was with the foot in dorsiflexion. All of these measurements were made by means of calipers, and the same level was obtained by means of superposition of the films over diffused strong light.

III. SOME DIFFICULTIES ENCOUNTERED IN INTERPRETATION OF THE ROENTGENOGRAMS OF THREE HUNDRED CASES

During the intensive study of the roentgenograms, from the point of view of roentgen interpretation, most interesting questions arose necessitating definite solutions before any classifications or any theories of mechanism could be determined. These questions came almost entirely from a lack of standardized technic. The 300 cases had been examined by four different roentgenologists. The technic of only one of them was known. Immediately the question arose: Does not this, or that, represent some variation from the real normal, produced by variations in the procedures of these different roentgenologists, rather than a pathologic condition?

Text-books on roentgenology describe certain more or less

standardized positions for examination of the ankle-joint. They point out anatomic landmarks above which to center the target of the tube. But none of them describe the variations in shadow which may occur: for instance, from increased or decreased target plate distance, or from variations produced by lateral shifting of the target, or by variations in posture of the joint. It would seem that opportunities for error are numerous. The very nature of hospital dressings -so different in the various institutions—constitutes one of the most prolific sources of error. The difficulty of exact centering through a plaster-of-Paris dressing or of an ankle encased in a fracture box is obvious.

The discussion which follows may seem to be far-fetched to some and futile to others. I am well aware of the conditions under which many busy roentgenologists work. I realize that a plate or film placed under an ankle with the target at any given distance above it, and the perpendicular assumed or guessed at, with a proper exposure technic will result in a so-called excellent negative. We admit that in a busy hospital service most of our patients were so examined. We admit that to the best of our knowledge no patients in any way afterward suffered, clinically, from such unstandardized examination at our hands. It is entirely probable that many clinicians can and will say, Why more? But I contend that in our study of these cases we were greatly hampered by such methods in arriving at definite conclusions. Gross lesions are usually apparent and easily diagnosed. The fine points in diagnosis are the difficult ones. It is only by exact methods that the science of diagnosis is advanced and the sum total of our knowledge of the subject increased.

The first difficulty that arose, the solution of which was of utmost importance, was the exact determination of just what on the roentgenogram determines diastasis of the lower tibiofibular juction. The normal ankle-joint was roentgenographed under all possible conditions, in an effort to study the possible variations in shadows of the normal due to changes in technic.

It was first examined at various target plate distances with the foot and target in the perpendicular plane; then the same ankle was roentgenographed with the leg in different angles of rotation and also with the foot in full dorsiflexion and plantar flexion, and again with the target deviated to either side from the perpendicular plane. A study of these results shows the necessity for the roentgenologist to decide on one target plate distance for all examinations; also for him to formulate or design some scheme for quickly securing in each case the same perpendicular plane of the ankle and target, especially in the anteroposterior view. A long upright rod with another at right angles, attached to its lower end, can readily be made and used for this purpose (goniometer).

From the results obtained on the roentgenograms showing the variations in target plate distance, it can be assumed that shadows on roentgenograms follow certain rules. Thus, as this distance decreases, all shadows proportionately increase provided there is no shift of the target in any direction, or any change in the position of the ankle, as, for instance, rotation of the limb. Thus the distance between the shadow of the line of the posterior tubercle on the fibular groove and that of the mesial margin of the fibula is increased if the target is moved nearer the plate, but likewise the width of the fibula and tibia seems to be proportionately increased. However, by no possible manipulation of the tube to either side up to 75 degrees from the perpendicular, or by inward or outward rotation of the leg to 80 degrees, can the shadow of the most lateral point of the anterior tubercle be made to pass to the median side of the shadow of the median border of the fibula. (It seemed that the above-mentioned angles were the limits of the possible errors that could have been made in the roentgenograms of our series.) Hence it was felt that we could safely say that whenever this did happen, tibiofibular diastasis was established. In fact, if the space between the lateral margin of the fibula and the lateral border of the anterior tubercle exceeds more than two-thirds of the width of the fibula, it is most probable that the

first degree of diastasis (Fig. 36) exists. This would seem to be certainly the case if the roentgenogram was made with the foot and ankle and tube in the absolute perpendicular plane (Figs. 49, 52 and 53).

FIG. 52.—Rotation outward, obliterating outline of the posterior tubercle. (Same happens in mesial deviation of the tube.) The anterior tubercle overlaps the lateral margin of the fibula.

Chaput[39] (1912) gave figures in millimeters showing variations of the "clear space," as he called it, between the line of the posterior tubercle and the mesial border of the fibula, and claimed that if this space exceeded more than 3 mm., or if the area of the overlapping shadows of fibula and anterior tibial tubercle

was less than 10 mm., diastasis was present. The objection to this as compared with the above method is obvious. Measurements on the roentgenogram vary greatly with changes in technic, target plate distance, etc., with age periods and in the different sexes. So a method establishing a means of estimation proportionate for each individual case is manifestly the best.

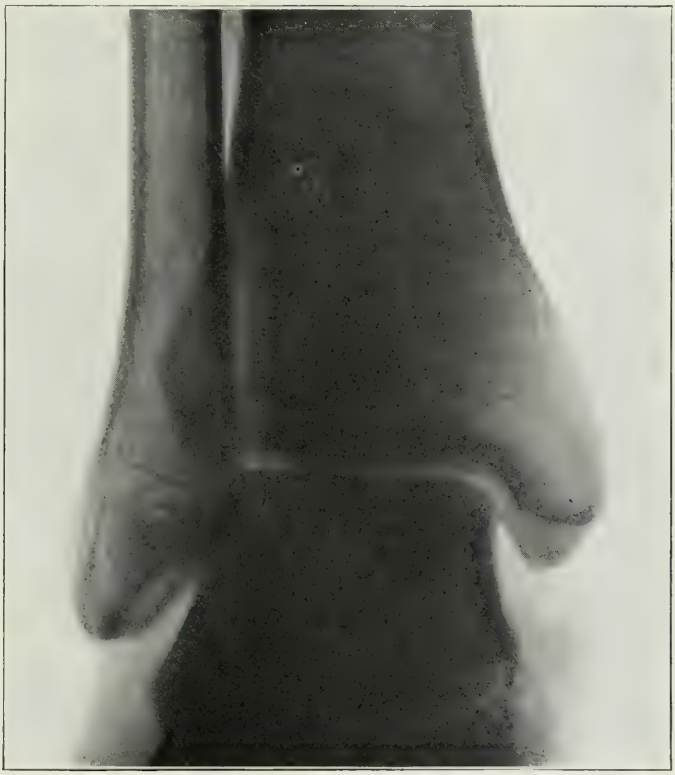


FIG. 53.—Rotation inward; very little change from the normal. (Same happens in lateral deviation of the tube.)

The larger the clear space between these bones, the greater the degree of diastasis. If there is additional roentgen evidence of a sprain fracture or detachment of the anterior tubercle, there is double assurance (Figs. 30 and 31). But here again

one must make due allowance for the fact that when the patient reaches the roentgenologist reduction often has been established. The clear space will then not be abnormally wide, and diastasis can only be presumed to have existed by the evidence of a sprain fracture or a fracture of the anterior tubercle. If these are not present, then no diagnosis of diastasis can be made by the roentgen ray. It seems possible that by a very complete study of normal ankles in both sexes and in all age periods, under identical conditions of technic, more absolute criteria can be established whereby this diagnosis can be made.

The clear space between the internal malleolus and the astragalus in the anteroposterior view is also a most interesting study. What determines the earliest or first degree of lateral displacement of the astragalus? This certainly is of the utmost importance in the determination of such displacement, particularly the so-called outward displacement. It was found that a 10 degree inward rotation of the leg increased this clear space, that the same amount of external rotation correspondingly decreased it; that variations in target plate distance proportionately increased or decreased it as the case might be, that tibial and fibular flexion of the foot both decreased it. So if a minute, careful diagnosis is to be made, the absolute perpendicular must be maintained. Had we had all roentgenograms of the 300 cases studied, made under identical technic, such diagnoses could have been made. We feel that quite a considerable degree of displacement had to be present, in fact so much that it would be apparent under any condition, any position of tube or ankle, before we could say definitely in the foregoing cases that a lateral dislocation existed. Probably also a better idea of rupture of the internal lateral ligament can be obtained by means of this clear space. Thus, when the internal malleolus is intact in the pure abduction fractures, this may afford an idea of whether or not the internal lateral ligament has ruptured. Here again faulty methods prevented exact conclusions, and this again emphasizes the possibility of study under standardized conditions.

Study of the fibular groove led to the definite conclusion that the anterior tubercle always casts the more lateral shadow, the posterior tubercle the more mesial. This was confirmed by placing pieces of lead of different sizes on the lips of the groove in a living subject and then roentgenographing the ankle. It was noted, in the negatives made with deviations of the tube and change in position of the ankle, above described, that in the perpendicular plane the line of the posterior tubercle was always clearly defined, that this clear definition persisted when the leg was rotated in or with lateral deviation of the target; but with the leg rotated out or with mesial deviation of the target, this line had a tendency to disappear, becoming merged with the dense shadow of the thicker middle portions of the tibia. It is apparent that such variations may materially interfere with the exact determination of just what constitutes the so-called posterior fragment. It may also interfere in the same way with recognition of the intermediate fragment. If this line remains intact, the posterior tubercle is not injured and the posterior inferior tibiofibular ligament is also probably not affected. It is possible that a deltoid shaped posterior marginal fragment broken from the tibia at a point mesial to and behind the posterior tubercle may be so displaced that its shadow overlaps the line of the posterior tubercle. The exact portion fractured can then only be determined by means of stereoscopic plates, and even then this is sometimes impossible. A fracture with such displacement was produced on a dried specimen, likewise another involving the posterior tubercle, so as to interfere with the line of the latter on the roentgen ray negative. In the anteroposterior view they were indistinguishable, both appearing to be fractures of the posterior tubercle. However, stereoscopic films showed quite plainly the character of the first, *i. e.*, a fragment displaced behind the tubercle and really not involving it.

In conclusion, this study has shown that while the roentgen diagnosis of the gross lesions of the ankle-joint is a comparatively easy matter for the trained roentgenologist, the finer points of

diagnosis so necessary to a thorough understanding of the mechanism are entirely dependent on more exact methods of technic than are often now employed.

In addition to the footnotes already given, the following, selected from more than 250 articles studied in the preparation of this paper, will be found of interest.

Bruns: Die Lehre von den Knochenbrüchen, Stuttgart, 1887, p. 57.

Farabeuf: Précis de manuel operatoire, Paris, 1909, p. 835.

Hamilton: Practical Treatise on Fractures and Dislocations, Philadelphia, 1860, pp. 443 and 685.

Messerer: Ueber Elasticität und Festigkeit der Menschlichen Knochen, Stuttgart, 1880, Plate 14, Figs. 7, 11 and 3.

Quénu: Bull. et mém. Soc. de chir. de Paris, 1906, xxxii, 943, Rev. de chir., 1907 xxxvi, 62,; Bull. et mém. Soc. de chir. de Paris, 1912, xxxviii, 1070; ibid., 1919, xlv, 1142.

Scudder: Treatment of Fractures, ed. 8, Philadelphia, 1915, p. 545.

Tillaux: Traité d'anatomie topographique, Paris, ed. 2, 1878, p. 1023; Gaz. d. hôp., 1886, lix, 89.

THE INFLUENCE OF PHYSICAL THERAPY IN REDUCING DISABILITY TIME IN FRACTURES OF THE LONG BONES

By JONATHAN M. WAINWRIGHT, M.D.
SCRANTON, PA.

In connection with the papers on fractures presented in the preceding pages, it has seemed justifiable to draw attention to the one definite point indicated in the title of this brief note. Experiences during and after the war have in a general way impressed us with the value of physical therapy. However, the enormous number of cases treated by these methods in army hospitals have no similar series for comparison. For this reason it has seemed profitable to prepare a table showing the disability times, in a fairly definite series of injuries, before and after the introduction of an adequate physical therapy department.

The appended table of "before" and "after" cases comprises about 125 in each group. Cases included are uncomplicated simple fractures only: that is, cases of fracture of the humerus with, for instance, musculospiral paralysis are not included, as they introduce an entirely new element. The figures are designed to show the average disability time of the ordinary case in each group of fractures. Further, the table only includes mine or railroad employees over fourteen years of age.

A study of this table gives very convincing mathematical evidence of the great economic value of physical therapy. In fractures of the clavicle and radius or ulna, where the disability times should not be long under any system, it has, of course, been impossible to cut the time to a large degree. But even

here the disability in each case has been reduced over 10 per cent. For the femur, tibia or fibula the reduction has naturally been larger, *i. e.*, over 25 per cent. The reduction for the lower leg has been practically one month, and for the femur, two months. Speaking in terms of cash only, this reduction has saved the corporation I represent about $4000 a year. And this limited fracture group is a small part of the total injuries of a year.

The most important deduction to be made from this study is the great advantage—almost the necessity—of the establishment of a physical therapy department in every large general hospital.

TABLE SHOWING REDUCTION OF DISABILITY TIMES IN FRACTURES OF THE LONG BONES AFTER ESTABLISHMENT OF PHYSICAL THERAPY GYMNASIUM AT MOSES TAYLOR HOSPITAL, SCRANTON, PA.

	Before Days disabled.	After Days disabled.	Saving in days.	Percentage. improvement in disability time.
Clavicle	67	59	8	12
Humerus	125	90	35	28
Radius, ulna, or both	76	64	12	16
Femur	239	180	59	25
Tibia, fibula, or both	121	92	29	24

Times are for uncomplicated simple fractures. Males over fourteen only, all mine or railroad employees.

DISCUSSION OF THE PAPERS ON FRACTURES

Dr. John B. Walker, New York:

In a study of our war injuries, it was found there were about 225,000 battle casualities, and of these there were 16,549 fractures of the long bones, 4053 being femurs.

In March, 1919, six months after the armistice, reports in the Office of the Surgeon-General indicated that a prognosis could be made that 76 per cent of fractures of the long bones would recover with satisfactory function. How widely this has varied is seen by the follow-up reports of the Bureau of War Risk over two years later, May 1, 1921, when it was found that 70 per cent of their fractures were suffering a disability of at least 20 per cent. Of 2306 femurs recorded in the Bureau of War Risk, 83 per cent are suffering with a disability of 20 per cent or more. Again as to the question of the duration of days of disability, reports in the Office of the Surgeon-General in 1919 estimated there would be an average duration of 300 days. Present reports of the Bureau of War Risk show the average has been over 450 days. These facts are quite startling in comparison with the earlier figures of prognosis.

Regarding mortality there has been great improvement. Of the 4053 fractures of the femur 1010 died, or 24 per cent. While in the Civil War, of the 6738 fractured femurs 3434 died, or 50 per cent.

When it is realized that two-thirds of all accident claims are based upon the consequences of fractures it becomes important that better efforts be made to bring down this loss of efficiency; that it is largely preventable has been shown by the splendid results of Depage, Pierson, Sinclair, Le Mesurier, Blake and others. Much better results will follow when surgeons give the same consideration to these cases which they now do to their abdominal cases.

Dr. Richard H. Harte, Philadelphia:

Incident to the general discussion on the subject of fractures, I will ask your indulgence if I may show a few lantern slides of fractures which are some of a large number that I collected with the assistance of our pathologist, Dr. Krumbhaar, while in charge of General Hospital No. 16, B. E. F., in France.

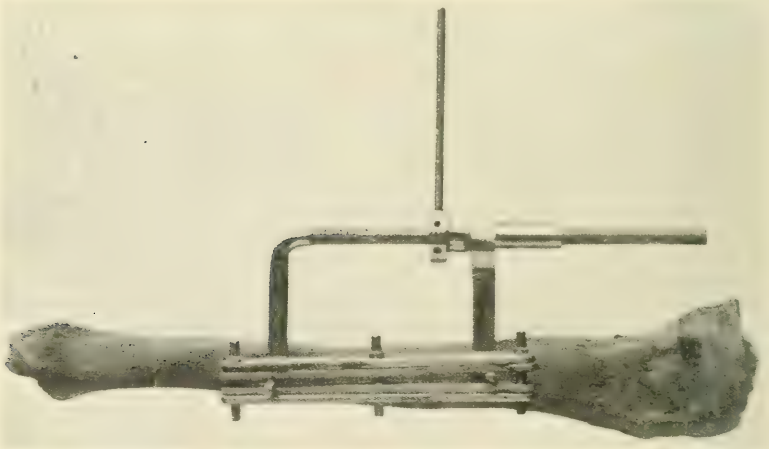
Inasmuch as "Tommy Atkins" has contributed all of the specimens, it seems only right and fitting that they should be first shown in the shadow of the Government for which they fought so hard and so successfully, giving their lives for liberty and now their bones to

science. The collection is a very large one indeed, and through the generosity of the British Government they are now well mounted, catalogued and in the museum of the College of Physicians of Philadelphia.

I cannot speak in too high terms of praise of the splendid coöperation and assistance that were rendered to us by the British Government all the time we were in France, and particularly by the Royal College of Surgeons in London in storing and preserving these specimens until such time that we could have them removed to their last resting-place at the College. The specimens represent every form of gunshot injury in its various destructiveness to the bones, so I need not go into detail in describing them, as they explain and tell their own story.

Dr. Leonard Freeman, Denver, Col.:

It may be of interest in this connection for me to exhibit an apparatus which I have devised for the treatment of overlapping fractures of the tibia. The apparatus consists (1) of two long bone-screws which are



inserted through button-hole incisions just large enough to admit the screws, at a distance from the site of the fracture, the screws penetrating both layers of bone so as to get a firm hold; (2) a turnbuckle, which is inserted between the two screws for the purpose of getting extension. (3) a bone-clamp, of an appropriate kind, to be clamped upon the screws so as to hold the fragments in place after extension. The turnbuckle

is then removed, leaving the bone-clamps to maintain the extension permanently.

The advantages of this sort of extension are: (1) Its ease of application without an open operation with its attendant dangers; (2) the ease of removal of the apparatus, which can be done without cutting and without a general anesthetic; (3) the absence of danger of infection (this has been well demonstrated by the Steinmann extension, which has been used so extensively during the war, and also by the number of times in which the Parkhill clamp has been used without infection); (4) the absence of any complicated apparatus which prevents the patient from moving; (5) the absence of any injury to the joints by pulling upon them, such as one gets in the ordinary traction apparatus; (6) there is also the advantage that the amount of extension can be accurately estimated, so that the surgeon feels satisfied that the bones are in place. The amount of extension is estimated by measurements of the opposite limb or by means of the *x*-ray.

Another advantage is that an apparatus of this kind can be applied where the skin has been injured, in compound fractures, and often when the skin is more or less diseased and so sensitive that adhesive plaster cannot be applied.

I think by inspection of the apparatus you will understand its working much better than I can explain it to you.

Dr. Howard Lilienthal, New York:

In compound fractures of the long bones, especially near the joints, where it is important to get an anatomically correct union, I can see absolutely no objection to the use of open rigid fixation of the fractured bones so as to hold them in perfect position. The wound should be laid wide open and the Carrel-Dakin treatment applied at once. The fixation may be by means of a Lane plate, with long screws extending out of the wound, the Freeman apparatus or the Smith bone-clamp. The important thing is to get immediate rigid fixation such as no external splint or apparatus can secure. The plate or other appliance can be removed at the end of from four to six weeks, when the tissues have been walled off by reparative processes and the danger of serious infection no longer exists. Then will be the time for external splinting.

In gunshot fractures a clamp is better than an appliance which requires the use of screws if the terrain where the fracture occurred harbors gas-forming organisms. I have used this method frequently and can testify to the tremendous relief which the patient feels as soon as the bones are rigidly fixed. When this method is applied early, while the parts are still mobile and before muscular spasm has deformed

the alignment, its application will be particularly easy. The method is also especially to be recommended when there is loss of bone.

I just want to correct one misapprehension: I said nothing about fractures into joints, I said fractures of the long bones near joints. I know there are a few men who disagree with me, but I am quite certain that the next few years will see me vindicated.

Dr. Emmet Rixford, San Francisco, Calif.:

I should like to add a little emphasis to what Dr. Ashhurst has said in reference to fractures at the ankle.

While not being quite ready to admit that pure abduction fracture at the ankle as described in the text-books does not occur, there is no question that in the great majority of instances in fractures classified as Pott's fracture the bone is broken largely by external rotation. One common and perfectly typical fracture is produced practically by external rotation alone. The anterior tibiofibular ligaments, being very stout, efficiently hold the lower end of the fibula in contact with the tibia. When external rotation occurs the long external malleolus is pressed outward and backward by the talus, the tensile forces are developed and the bone breaks, the fissure starting at the inferior margin of the anterior tibiofibular ligaments. The fissure is propagated upward and backward in a more or less spiral direction, emerging at the posterior border of the fibula above the posterior tibiofibular ligaments. It therefore follows that the upper fragment of the fibula remains attached to the tibia by the anterior tibiofibular ligaments, while the lower fragment remains attached to the lower end of the tibia by the posterior ligaments. In a large proportion of cases, either as the effect of complicating longitudinal thrust, but sometimes doubtless due to a continuance of the rotation, a portion of the posterior lip of the tibia is torn away and remains attached to the lower fragment of the fibula. This fracture may be exceedingly difficult to maintain in proper adjustment, and is especially so if the lower fragment of the tibia is of any considerable size.

Dr. Joseph A. Blake, New York:

A discussion is usually interesting when there is a difference of opinion, and I believe it always makes it more interesting when we have decided expression of opinion for or against.

In regard to the question that Dr. Lilienthal brought up as to immediate fixation of compound fractures: These fractures, of course, in civil life are entirely different from war fractures. War fractures are usually contaminated and always contaminated when caused by high explosives. Not to speak of individual cases, but of cases by the

thousands, internal fixation of gunshot fractures is bad practice. This was proven by repeated observations. The ordinary compound fracture in civil life is compounded from within, usually is not contaminated as in gunshot fractures and frequently escapes infection, therefore internal fixation may be permissible.

As to the primary treatment to be applied, "dèbridement," as we call it in America, "epluchage" in France, *i. e.* paring of the wound, is practised. This is necessary and all contaminated parts are cut away. Then if one knows how to properly treat fractures by apparatus there is no fear of getting bad results and no need of using internal fixation. The objections to internal fixation are that if a fracture becomes infected the foreign material is distinctly disadvantageous and may cause a prolongation of the infection, an absorption of bone and possibly non-union, or it may lead even to amputation.

The discussion of the subject before us is almost impossible because the subject is too great. Two or three points I should like to bring out. In the first place we have had an interesting comparison of the methods of grafting. In my experience, in doing a great number of grafts and observing a greater number, I believe that the inlay graft should be restricted to the larger bones, and I believe that for the smaller bones we should use osteoperiosteal grafts, that is, grafts made by shaving the bone off with the periosteum. As Dr. McWilliams has shown, this method has a high percentage of good results and does not remove bone. In the case of the small bones, such as the radius and ulna, the inlay graft should not be used. Osteoperiosteal grafting as carried out in the French hospitals has been extremely successful, and I believe its superiority to other methods is due to its adding bone instead of possibly causing the death and loss of large portions of bone.

Among the ex-service men coming to the Polyclinic Hospital in New York there have been a great many cases of non-union that had been treated by bone grafts, and among them I have yet to see a successful inlay graft. In nearly every one the inlay graft had to be removed.

This recalls to my mind a recent case which illustrates both the question of internal fixation and of inlay grafting. This patient, five years ago, suffered a fracture of the femur and was treated by internal fixation with bone plates. There was infection, removal of the plates and continued non-union. Finally, after four years, introduction of an inlay graft; removal of this graft about two months ago on account of continuous suppuration, the graft being found to be lying in a bed of pus. At present there is still non-union, and in my opinion amputation is the only thing to be done, but the patient objects to this.

In closing I beg to state that we have with us today the gentleman who had the most outstanding and best fracture service in the French

Army during the war, the man who successfully led the way in excision of fractures and particularly in excision of joints, and it seems to me we should have some discussion from him, Dr. Leriché.

Dr. John H. Gibbon, Philadelphia:

It seems to me that this question of the treatment of fractures, which we are so anxious to advance generally in this country, should first be taken up from the point of view of the medical student. Interns come to us with practically no knowledge of the anatomy which they should have in order properly to understand reduction, retention and restoration of function.

We have had presented to us today very unusual types of fractures, all of which require operation, but we must remember that there are hundreds of fractures which will be treated by the general practitioner. I think that the medical schools are at fault in teaching fractures, or I should say in not teaching fractures. I would suggest that in teaching fractures to medical students 99 per cent of the time should be spent in the discussion of the cause of fractures, the cause of the deformity of the individual fractures, methods of overcoming it, the restoration of function, and 1 per cent devoted to operative treatment.

In regard to the operative treatment there are certain things that experience has shown which we must recognize when we come to operate on fractures. In the first place an operation upon a bad fracture will not shorten the time of the patient's treatment. If infection occurs it may be prolonged and will probably necessitate further operations to correct it.

In the next place operation with absolute immobilization of the fragments, which is our object in operating, is a cause of delayed union, sometimes of non-union. The introduction of foreign bodies, especially plates, is often the cause of refracture.

If we look at the report of the British Medical Association on Fractures we find that the ultimate functional results in the cases treated by mobilization (Lucas Championnière and Mennel) are often better than those which have followed the operative treatment or prolonged fixation.

I do not agree with Dr. Lilienthal's view that compound fractures involving the joints should be subjected to operation unless a fracture is rendered compound by the penetration of a foreign body. If penetration of the soft tissues is caused by the bone being thrust through, then I believe that all the parts should be painted with iodin and the fracture reduced with no attempt made to fix the fragments or drain the wound. I have seen a large number of compound Pott's fractures in which the articular surface of the tibia protruded and in which

prompt healing and good functional results followed the simple method just mentioned. I am convinced that a large percentage of recurrences follow operative interference.

I agree absolutely with the remarks made by Dr. Blake. Another point which I should like to make in regard to the operative treatment of fractures is that this treatment is resorted to entirely too often in the case of children. *X*-ray plates taken years after what seem to be bad results will demonstrate to a surprising degree what Nature is able to do to restore contour and function in these growing bones. In our great operative zeal I am afraid we have overlooked the fact that function to the majority of our patients is far more valuable than perfect alignment. I realize, of course, that perfect reduction may give a perfect restoration of function, but we must do nothing to obtain it which may sacrifice the ultimate functional result. My remarks apply to compound fractures whether near or in the joint.

Dr. A. A. Law, Minneapolis, Minn.:

I wish to register my appreciation of Dr. Lewis's courage. I speak particularly of his temerity in bone-grafting in the presence of infection. My interest was stimulated in this subject in 1913, and in 1915, in Rochester, I read a paper entitled "Autografts in Infected Fields." This was published in 1916. In this paper I reported several cases of the successful transplantation of fascia, fat and bone, which survived in the presence of chronically infected fields.

We reported a long series of laboratory research operations where autografts of fat, fascia and bone were transplanted into acutely and chronically infected fields in dogs and rabbits.

The autografts in the presence of the acute infections were lost, while in each instance the grafts introduced into the chronically infected field survived either in part or as a whole.

We were convinced after three years of clinical and laboratory research that it was feasible, and in selected cases proper, to make autogenous transplants in the presence of chronic infection, for after long infection a degree of immunity is established to that infection. This common immunity is shared by all the cells and fluids of the infected economy, therefore the transplants have already established the same immunity as the field into which they are transplanted.

It is to be hoped that some day methods may be devised which will enable us to measure the degree of immunity and thereby give us a definite justification for autotransplantations in infected fields.

Dr. Charles L. Scudder, Boston:

If I may continue the subject of Dr. Estes's report: It is a fact that surgeons throughout the country are not interested in fractures. One

evidence of this lack of interest is the difficulty that the Fracture Committee, of which Dr. Estes is chairman, has had in securing satisfactory data for the report. I believe we all agree that we need better results. The solution of the difficulty is largely in two directions: (1) In the adequate instruction of undergraduate medical students, arousing an interest in them to take up the study of traumatic surgery, and (2) in the inauguration of proper graduate instruction through our graduate schools.

Dr. John A. Hartwell, New York:

I only want to add that one of the purposes of presenting this discussion is to call attention to the enormous opportunities of treating fractures that occur in some hospitals. At Bellevue Hospital, since we have been taking care of them, we have had so many that we are employing two or three men constantly looking after fractures. We began to do this before the war, but the war gave us a special incentive to do it.

With reference to our experience with the open operation, we have practically abandoned it unless in extreme cases and where it is impossible to reduce it in any other way. We have abandoned it for the reasons given by Drs. Blake and Gibbon. Even in severe fractures if we spend sufficient time we can get exceedingly good results. As to the anatomic results we have not succeeded in obtaining as good position of the bones as we want, but we have great confidence that with increased effort we shall be able to do so, so that we will add to the functional result a very good anatomic result, and we will not be faced by the disaster that has been pointed out here, of which we have had a large number.

At Bellevue Hospital, like all municipal hospitals, we have a large number of cases that fail to do well in other places. Over a long period we have been receiving positive evidence of the great danger of operating upon fractures. Many of them have been operated upon by able men, and we have reached the conclusion that an open operation on a fracture of one of the large bones is a very serious undertaking, and we will keep away from it as long as we can find any other method of getting a good functional result.

Dr. Emmet Rixford, San Francisco, Calif.:

I just want to say one word more to correct an impression that I inadvertently gave in reading my paper, when I said that I thought that all spiral fractures of the long bones would best be relieved by open operation. In my paper it is written, "Children are specifically excepted."

For a number of reasons, not only those stated by Dr. Gibbon but also in children, they are usually oblique. The bone undergoes a considerable degree of torsion before it breaks and therefore the spiral fracture is much more oblique.

In my paper I also attempted to submit a definite technic and would emphasize as a part of that the great advantage to the surgeon of having the active coöperation of an intelligent physiotherapeutist, someone who can use his electric apparatus and massage for maintaining the tone and nutrition of the muscles and of the limb until such time as function is restored. I think the earlier functional demand is made under suitable conditions the more rapidly the bones will unite.

OLD OS CALCIS FRACTURES*

By FRED J. COTTON, M.D.
BOSTON, MASS.

Os calcis fractures, not common, scarcely seen except as the result of industrial accidents, are of interest because they give so large a percentage of cripples, and because these cripples are strong men, as a rule, in youth or vigorous middle age.

Years ago[1] I went into the question of the treatment of the fresh os calcis fractures, basing recommendations on a serious investigation of a long series of old cases at the City Hospital, and on a recognition, by us apparently for the first time, of the essentials of the injury and the real causes of disability.

Neither in the cases in this paper, nor in those of a later one,[2] nor in the cases collected by the Industrial Accident Board of Massachusetts[3] have we acquired data on which to base any accurate percentage statement of disability.

There are, under ordinary treatment, a few cases (without much original displacement) that recover with good or fair feet—these are cases mainly of little displacement; a large proportion, certainly much more than half the total, are partly disabled and handicapped in their work; total disability for real work seems to be the fate of something like one-third to one-half the cases.

There is, I think, nothing to add to what I have already written as to fresh cases in the above-cited articles and in a book[4] on fractures, as to lesions or treatment, save for the addition of later cases. Today I should perhaps lay more stress on remodeling, less on "impaction," for I suspect that the impaction resulting is not mechanically first-rate as a rule.

* Read by title.

This remodeling or impaction method has been adopted by a good many surgeons with good results, I think, as a rule. My own cases have been not perfect, of course, but satisfactory, and I think there have been none with serious permanent disability. But, while I see fewer fresh cases each year,[5] on the other hand I see, of late, more and more a number of the cripples untreated —very often diagnosed as sprained ankles in the early weeks. These men, disabled, out of work in several instances for a year or two years, have been referred to me by insurers as a rule, with the idea of returning them to industry. They have usually been baked and massaged with no benefit (except to the baker): the "bakee," if one may so call him, still limps, and keeps on with his compensation under the accident laws. The problem is not new, or easy. My earlier attempts were rewarded by improvement, not by cures.

I had worked with the idea of clearing away excess bone, on the outer side, removing spurs, and limbering up joints by manipulation.

Prince, of Rochester, N. Y., has done arthrodesis of the posterior calcaneo-astragaloid joint to relieve strain. No one else seems to have done anything in particular.

In the last two years I have been beginning to get real results—good enough to feel that we have something approaching a solution of the problem. Wherefore, this paper was prepared.

The cases run curiously similar as a rule in their main features. In the first place, they all have the calcis a little short from front to back, a bit flattened, often more than a bit, on the sole. Beyond a little loss of "spring," this shortening seems to make little difference. 2. Rarely there is sharp outward deviation of the whole heel. This entails inevitable flat-foot, not to be relieved by supports. The deformity must be corrected by the Gleich operation, of which more later.

3. Spurs on the plantar face of the calcis are not uncommon. If tender, as they usually are, they must come out.

4. In every crippled case that I have seen, the outward broadening due to the shoving outward of the peroneal plate of bone

and to bone-growth behind it, has been a source of trouble. Sometimes the external malleolus hits the exuberant bone or is half buried in it: sometimes it hits only in extension or only in flexion: sometimes it merely pinches the peroneal tendons and their sheath; but, invariably it is a factor in the pain and disability.

How considerable this mass often is will be seen from some of the *x*-rays taken from above and behind—a rather useful even if an unusual view.

5. The loss of some part of the lateral motion is constant; loss of all lateral motion not rare. As a rule, this limited motion is, at its limits, painful, and is always a bit disabling because of resultant clumsiness. This loss of motion results from what we may call clogging of the posterior calcaneo-astragaloid joint either from fracture across it or from fracture displacing the unbroken joint-surfaces or shortening the slide; or from new bone heaped up roundabout the malleolus or in front. Practically all motions of the foot laterally, remember, depend on free motion in the two joints on the under surface of the astragalus, both joints articulating with the os calcis. With even only the posterior joint locked, the lateral mobility of the foot is gone.

Now, as to the repair of these various "proximate causes of disability." (1) Shortened and flattened heel; not to be touched, save when combined with outward displacement. (2) Outward deviation; calls for the "Gleich" operation, an operation devised for the cure of flat-foot—a cross-section of the calcis done behind the posterior joint of the os calcis with the astragalus. The original description specified a Gigli saw—I have preferred a thin chisel, cutting on a slant slightly down and forward, forward and inward. The calcis so cut, it is then easy to displace the heel as one will—with ragged chiselled surfaces, no other fixation than the plaster is needed. This operation I have done twice; it is a perfectly simple and straightforward maneuver. (3) Spurs; sometimes they are spurs of an original fragment, sometimes perhaps newly grown. They are to be removed, just like the common osteophytic spurs, through an incision at

the side, well above the thick skin of the heel, and they are to be removed very liberally, as are all os calcis spurs. (4) and (5) These are in fact one problem. The fracture is a crushing fracture with irregular cleavage lines. The result of the usual treatment advocated, which is by way of being no treatment at all, is the formation of a broad, shapeless bone, which has lost all chance of rocking under the astragalus, which has spread so far out and up under the outer malleolus as to give crippling pain from bone contact beside the loss of motion. The failure of a good many attempts to remedy this, including my own earlier efforts, was due to our failure to appreciate the mass of this bone growth, and its great total bulk, and to take account of the fact, well enough known really, that the tarsal bones repair their injuries with unusually massive bone replacement.[6] One must do far more than at first seems reasonable.

The key to this whole question is the doing of enough surgery. It is like the man who "shaves a day ahead;" one must do much more than to remove the presenting excess of bone. Latterly, I have doubled the bone removed, have dug deep out and wide under the peroneal tendons, and then laid them back under pressure. More than that, I have cut ruthlessly across the joint line between astragalus and calcis, often leaving what seems not half the joint.

The operation, as developed, is a skin incision, curved down and forward beneath the external malleolus—a stripping up and laying forward and upward of a flap, including the peroneal tendons in their sheath, turning up with them the periosteum and with it the cortical layer of bone; then comes a thorough clearing away of all the excess bone, deep below the cortical level, leaving a saucer-line crater of bone. In doing this clearing away one cuts across the posterior astragalo-calcaneal joint regardless of ligaments and of joint capsule. (See Fig. 1.) Then, forced manipulation in rotation and in abduction and adduction of the foot complete the clearing of any obstacle to motion from bone, from adhesions or from scar. At this stage, after reduction, motion should be near normal and unimpeded by adhesions

or by excess bone. After the clearing away of excess bone the periosteal bone flap, carrying the tendons, is laid down into the crater from which the discarded bone was removed. Loose suturing completes the open operation.

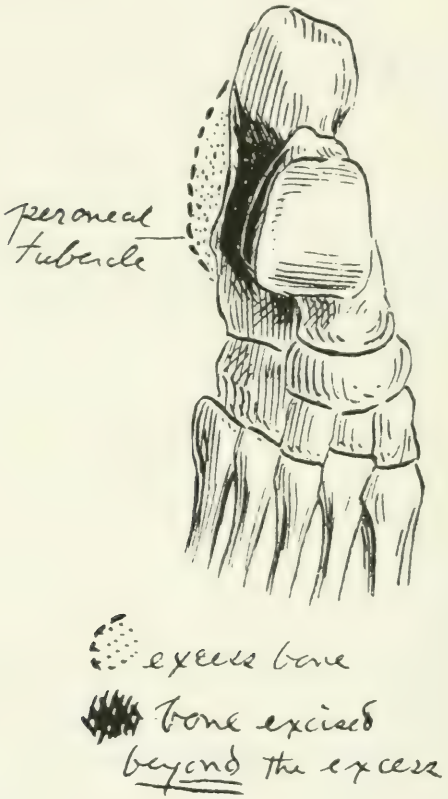


FIG. 1

Plaster is applied in neutral position, and without regard for right-angled flexion. One has never to fear loss of ankle-joint motion in these cases; it simply does not happen; and I have long preferred a position of moderate plantar flexion to the alternative of tenotomy of the T. Achillis, which not uncommonly leads to a rather long-continued atrophy of the calf muscles.[7]

Pressure from outside is applied to the case to keep the deep flap in place under the malleolus in the crater provided by the operation. After two weeks the plaster is cut and guarded motion is begun.

After four weeks one may allow a little weight bearing—cautiously, and motion is emphasized.

At six weeks weight-bearing is allowed; still with some caution, gradually increasing the weight borne, steadily emphasizing the regaining of motion by voluntary exercise, aided by stretching in the hands of the masseuse in case of the laggards.

In the less likely cases arch plates are used, temporarily—either of the usual type, or of the "rocker" plate (the "Whitman" sort)—worn until the foot comes back to useful function.

For the cases so treated, one may claim actually normal function in one; approximately normal, one may say practically normal in six more of the total of nine operated on.[8] One case, an elderly man, not robust, still has some arthritis tenderness, mainly of the front part of the foot, that interferes a bit with his work.

One case I write down a failure—probably because no one here understands Russian, and he speaks only Russian of some sort. Therefore the routine of exercises could not be impressed on him effectively. Perhaps we mistook his smile for comprehension. I suspect he, too, will later return to work, but for the moment he is written down a failure. The others are cured. Of the two treated as fresh lesions, both are at work, near normal in function. Three, not operated on, have come back to near normal under persistent treatment. Two are too recent to report on.

The case records follow:

Case I. T. McN., aged thirty-six years. Carpenter's helper.

Accident: October 24, 1917, fell squarely on both feet. Eleven weeks in bed. One month on crutches; then two canes; able to walk without canes about March, 1918. Referred by London Guarantee and Accident Company, Limited (Figs. 2 and 3).

Pain and weakness about ankle of right foot. Marked disability.

Left foot showed on examination by F. J. C., June 10, 1919, a "rigid flat-foot" not of extreme degree, nothing else.

Right foot showed "very evident os calcis fracture with marked thickening below the external malleolus, with definite broadening of the heel seen from behind with a spur on both sides so large as to be easily felt with the fingers. The foot not much flattened and the heel is not turned outward. No lateral motion. Fair ankle motion." This man has had a fracture of the right os

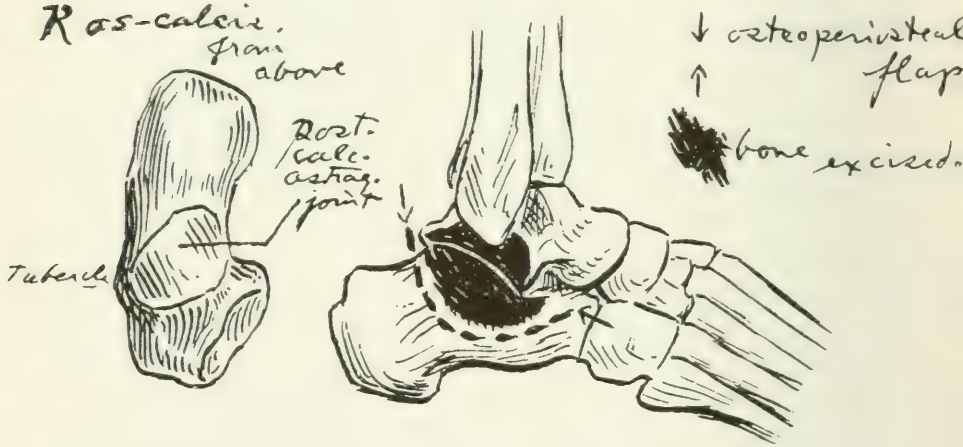


FIG. 2

calcis with marked spur projection into the sole. This is the tender point. There is also some tenderness below the outer malleolus and some soreness running down the foot from this point. No swelling. The general condition and ability to use this foot have become, under much massage, much better than one would expect from such findings.

Operation proposed to clear the spur and to remove excess bone on the outer side.

It is worth noting that an astragalectomy operation had been advised on this case less than a month before I saw him. One is

at a loss to see how an astragalectomy could help; certainly it would cripple. I have yet to see a really good working result after astragalectomy—and I have seen not a few.

June 3, 1919, the usual operation was done, including also in this case the removal of a sharp spur. (See Fig. 3.)

Convalescence was as usual, but slow, which is hardly unusual. It was necessary to fuss with a plate for a time, with anterior arch pads also.[9] He also complained of toe-contractures for a time.

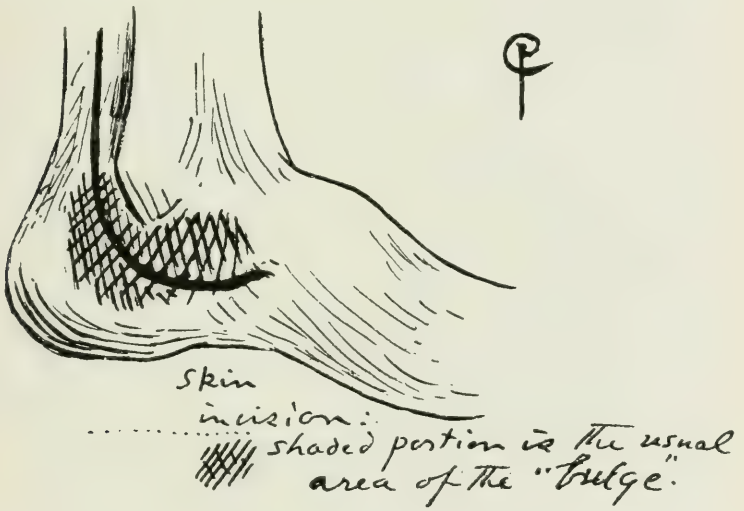


FIG. 3

Some time (I have not the date) early in 1920, he went back to and has stayed back at work, with serviceable recovery. Disability before operation one year and eight months. After operation, under nine months.

Moreover, this result was impeded a bit by the spastic flat-foot on the other side—treated, but yielding to treatment slowly.

The foot operated on showed the usual return of well over half the lateral mobility. There was entire, though slow, cure of the sensitiveness from spur pressure.

Case II. T. McN., aged thirty-three years. Lineman.

May 25, 1918, fell fifteen feet, landing on left heel. Other foot unhurt. No ether for reduction or pseudoreduction. "Cast" for seven weeks. Crutches up to about June, 1919. Referred by the London Guarantee and Accident Company, Limited.

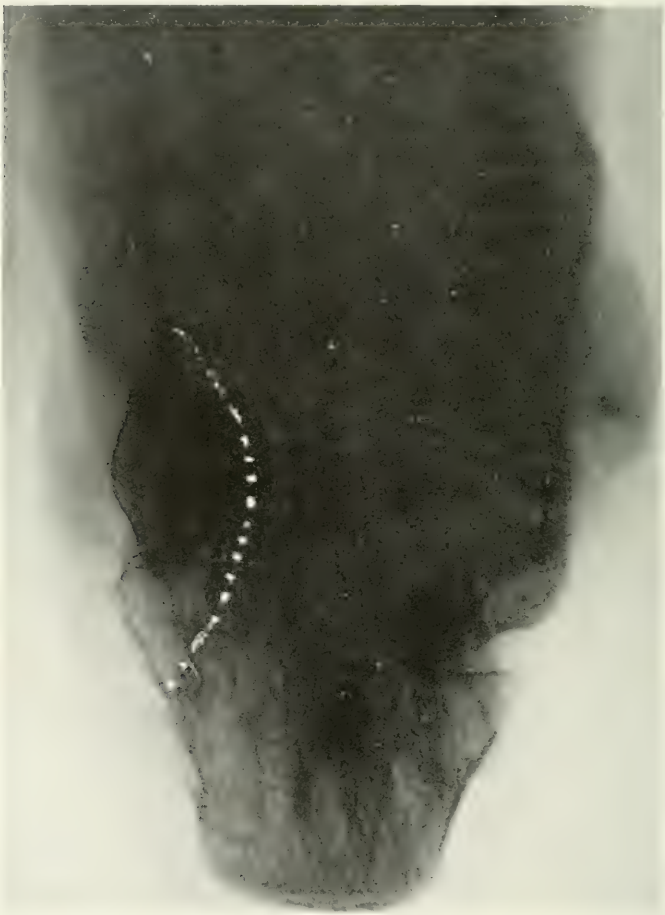


Fig. 4.—Case I (from above and behind). Huge broadening of heel, with the peroneal plate shoved up squarely against the external malleolus. White dotted line shows roughly the amount of bone removed at operation.

Seen by F. J. C., August 6, 1919. Previous to this I had had sent to me plates of both his feet, showing, besides the deformity in one, an appalling "traumatic arthritis" picture of osteoporosis in both feet. Examination of the feet themselves, not hot or tender or swollen or spastic, made it evident that this was a result not of an essential arthritic process, but of the fifteen months of utter disuse. Examination showed on the right only an old flat-foot, strained from having more than its share of labor imposed on it.

"The left foot, on the other hand, shows a condition which I think will have to be remedied by operation. The os calcis has been fractured and there has been marked displacement of the back portion of the bone (which includes the bearing surface of the heel) outward rather than upward. The flattening of the foot by bone displacement is less than usual, the thickening of the foot no more than average in these cases, but this outward displacement is very extreme and I think without question that it is too great to be remedied effectively without an operation."

The operation proposed was the Gleich operation, namely, a section of the astragalus behind the inner vessels and tendons, down and in, forward and inward.

This was done October 27, 1919, and with the Gleich operation the excess bone below the external malleolus was removed in the usual fashion (Fig. 4). He was in the hospital for just a month. There was the usual slow recovery and gradually increasing use. At about a year[10] he went back to work, and, according to a friend he referred to me lately, is still at work.

Disability, original, before operation, seventeen months. After operation, not quite a year. Result almost 100 per cent.

Case III. W. T., aged twenty-one years. Sprinkler fitter's helper. On November 14, 1918, fell forty feet. Treated at City Hospital, and about January, 1919, came under treatment of Dr. A. T. Cornwall. Seen by F. J. C., May 26, 1919, for the Accident Board. Reported as a result better than usual, but with broadening and much thickening of bone on the outer side. Lateral

motion much better than usual, but some pain on the outer side in even moderate pronation. Trouble only occasionally, but it involves an uncertainty that bars him, from fear of accident. Sent by the U. S. Casualty Company for operation.

Operation August, 1919. The usual operation. He did well, but slowly. Motion excellent and no longer any "catching" on the outer side. He was fitted to a plate with relief and complained only of occasional pain down the outer side of the foot. The foot is practically normal in appearance and nearly normal in motion. He worked a month in the fall of 1921. Later complained of contractured "hammer toes" and was given treatment to stretch them. Redischarged as practically normal in February, 1921.

This is in fact a perfectly good cure. Personal disinclination and an unfavorable labor market just now are the key to the persistent disability.

Case IV. A. F. G., aged forty-one years. Accident July 21, 1919. Fracture right os calcis; treatment persisted in without much improvement. Seen by F. J. C., May 21, 1920; referred by the U. S. Mutual Liability Insurance Company. He showed the usual picture with total loss of lateral mobility, with much pain from apparent contact of malleolus and calcis, and also unusual inability to come up on his toes because of contact of the malleolus with a mass of thickened bone far back (Fig. 5).

He was particularly annoyed because as sole survivor of a gun-squad of the Cameronian Highlanders, and shot-up at that, he felt it beneath his dignity to succumb to so unexciting a lesion.

Operation June 1, 1920. The usual operation but with a very liberal removal of bone. In hospital twelve days. Plates and massage as usual. Last seen September 23, 1920, at which time there was still some broadening but almost full mobility. He then demonstrated his ability to jump up in air and crack his heels together twice—not equal to his previous normal of three times, but fair enough. He then went back on his job, and so far as I know is still there.

Case V. F. McJ. Accident September 9, 1919. Fell thirty to forty feet on his heels. City Hospital. Plaster six weeks; later electrical treatment with Dr. Frank Gardner. Report of examination April 23, 1920, showed typical condition, thickening, pain on standing or walking, etc. Further treatment instituted. Seen by F. J. C. Referred by Maryland Casualty Company. Usual flattening, broadening, thickening to the outer side, loss of most of the lateral motion (Fig. 6).

Fig. 5.—Case VII. Flattened heel. Thickening obvious at X. Spur on plantar surface from projecting fragment.

Operation September 27, 1920. Treatment and recovery as usual.

Improved rather quickly but showed some tendency to pronate; relieved by plates and by juggling the shoes. Last seen April, 1921. No evidence of disability at all, though lateral motion not to full normal limits.

Case VI. B. O., window washer. Accident November 8, 1919. Fell two stories. Referred by Dr. Frank Gardner for the Maryland Casualty Company. Seen by F. J. C., August 20, 1920. Showed clear evidence of double os calcis fracture with little displacement, not much thickening, and good motion on the right.

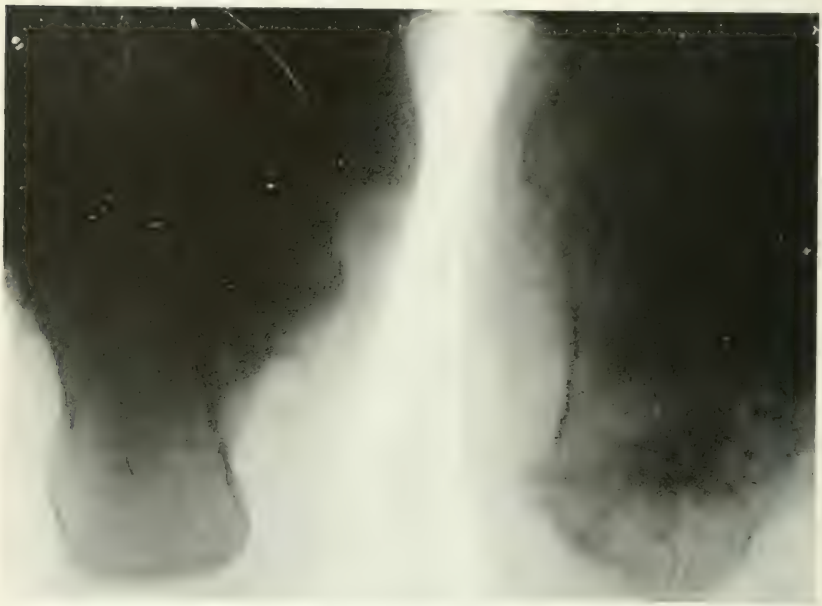


Fig. 6.—Case II. Fifty-two days after a "Gleich" operation. Heel now properly under the foot. Still much thickening, despite considerable removal of bone. Here, as always, tarsal bone repairs in excess.

On the left extreme thickening, "chocking up" against the fibula, not only laterally, but also at the front, with almost no lateral motion. Disability to correspond.

Operation August 30, 1920. Usual technic. Hospital one month, no sepsis, but the serous leakage (almost always present for a week or ten days in these cases) persisted a bit longer in this instance, perhaps because an unusual amount of excess bone was removed.

Returned to Dr. Gardner's care. Recovery very satisfactory, and final function, March, 1921, practically normal as to shape and motion. No complaint save of occasional pain on outer side of foot running forward to the last two toes.[11] Seen June 13, 1921; still has some discomfort, particularly in rising on toes, and is the first case I have seen with limited dorsal flexion. Supination about half; other motions normal.

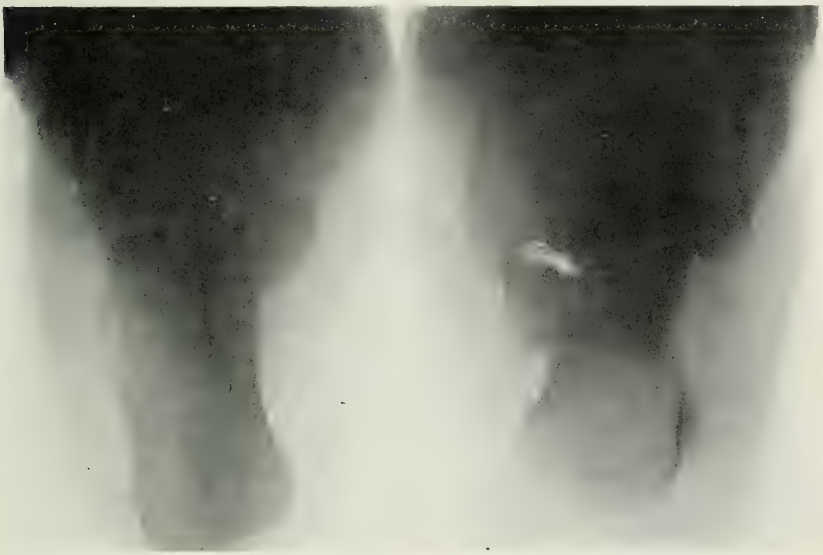


FIG. 7.—Case IV. Right heel shortened; not much outward thickening. Main disability here was loss of lateral motion—restored by operation.

CASE VII. D. P. Seen by F. J. C., August 13, 1920. Man of fifty-six years, not of vigorous type. Injury four months previous; fell fifteen feet. Referred by Dr. S. P. Strickland, of Waltham, Mass., for the Federal Mutual Liability Insurance Company.

Showed fracture of left os calcis with lateral motion entirely gone, crippling pain to the outer side on walking, enormous thickening to the outer side, confirmed by taking *x*-rays from behind and above. (See Fig. 7.) The *x*-ray showed less osteo-

porosis than the case-history suggested, but did show a discomforting degree of arteriosclerosis of the vessels about the ankle. Operation advised, nevertheless.

Operation, August 24, 1920. Result: September 17, entire removal of the conspicuous deformity, no swelling, slight tenderness, already about one-third normal motion. Later there were

FIG. 8.—Case V. Left os calcis broadened; outer plate displaced far outward.

arthritic symptoms and static troubles with the anterior arch, etc. He started in at work the first of the year 1921, overworked a bit, and, despite shoeing and a plate, still has a foot that tires and swells a little and does not allow him full working time, but he has his motion, and none of the symptoms (except a bit of nerve pain as in Case III) are at all referable to the region of the os calcis. They are anterior arch pains, and swelling of

the whole leg on standing, due to a circulation not young and none too good.

CASE VIII. H. K., May 26, 1919, fell forty to fifty feet, and broke his back (compression fracture first lumbar, without cord-lesions, and practically without nerve-root symptoms). Also

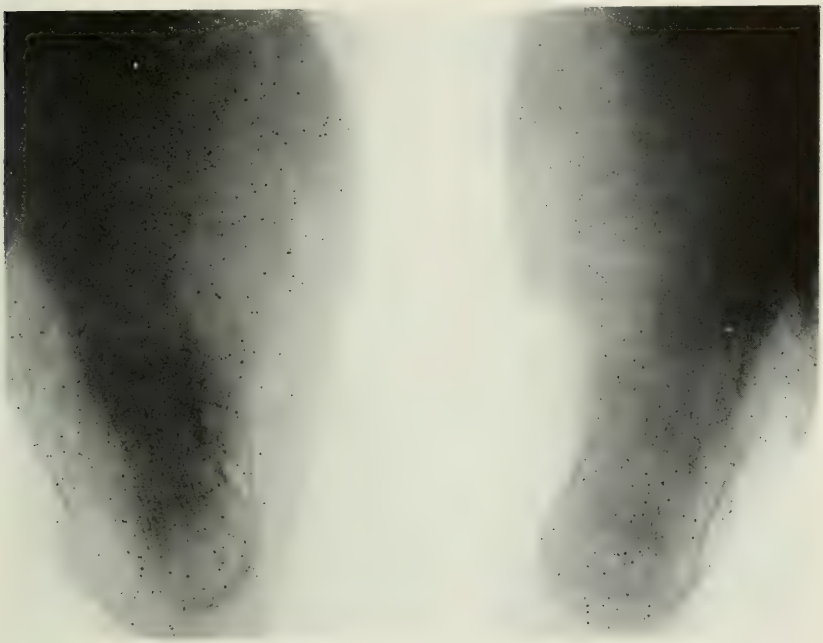


FIG. 9.—Case VII. Much broadening, excessive thickening below external malleolus. Marked osteoporosis.

he broke his right foot, but the diagnosis of Pott's fracture was made.

Accordingly he was sent in by the U. S. Mutual Liability Insurance Company. Examined by F. J. C., on July 27, 1920; the treated back was nearly well. The untreated foot showed a typical os calcis fracture with the usual total loss of lateral motion with lameness on the outer side, with massive bone

thickening beneath the external malleolus and with tenderness in the sole as well, due to spur points projecting downward.

Operation July 30, 1920. Usual technic with removal of much bone at the side and of a spur also, projecting down into the sole toward the inner side. He did very well indeed for a time, during his month in hospital; after this less well, owing in part

FIG. 10.—Case IX. Moderate deformity only. The "spur" on the inner tuberosity is real, and corresponds with the tender point in the sole, but is a new growth and not the usual spur from projection of a fragment, as seen for instance in Fig. 3.

to his being a Russian, speaking an unattainable language, unable to understand very clearly about exercise and use.

This case lost his lateral motion to a considerable extent, and when last seen in November, 1920, while he was much better than before operation, with less pain and more motion, he was not fit for heavy labor. Not all this unfitness was due to the ankle,

the back also had to be reckoned with, but the result of the os calcis operation in this case was less happy than in the others.

The operation worked well enough, the after-treatment did not quite work out.

CASE IX. P. S. Examination April 28, 1921, for the Federal Mutual Liability Insurance Company. A year ago, June 11, 1920, he fell and fractured os calcis, right and left. Has been under treatment—plates, etc. Has almost no limp, and on detailed inspection little save moderate thickening on the left. On the right there is more thickening and there is pain when he tries to come up on his toes, apparently from contact of the thickened bone with the external malleolus. The bone thickening toward the back is unusually heavy. Also there is a tender point below the internal tuberosity of the calcis.

There is little tendency to pronation on either side and motion both right and left is good—perhaps one-third normal (Fig. 8).

This case looks well enough, but, apparently owing to the bony thickening on the left side, he has been in the discard industrially for over a year. The *x*-ray shows only moderate deformity. He is to be operated on in the near future.

CASE X. H. C., aged thirty-four years. Painter. Broke heel two years ago in fall from staging. Has gone on under more or less treatment without essential change. He has the usual thickening, lateral motion not over a quarter in supination, and while he has no pain if he loafs, he is good for about two miles only, then has pain and stiffness. Is also stiff of mornings. Pain all referred to region below external malleolus where the thickening is heavy. He has backed and filled for two months, but, having decided he is useless as things lie, he is to come up for operation June 15.[12]

It is only fair to give the other cases—non-operative.

CASE XI. M. B., aged twenty-nine years. Navy yard electrician, referred by Major E. K. Sprague, of the Marine Hospital of the Public Health Service of the Treasury Department; had had a double

calcis fracture. He fell January 8, 1920, a distance of eighteen feet. Treated without reduction for seven and a half weeks. Then had some sort of correction under ether and a fortnight more of "cast" treatment. Has walked since then.

Examined, F. J. C., June 11, 1920. Showed clearly enough fracture of os calcis, right and left. On the right it was not so bad—motion fair, pain little, bony thickening reasonable. On the left, less motion and massive thickening to the outer side, but little pain. This case looked hopeful.

Given left inside heel, right one-quarter inch, left one-sixth inch, and shown how to do rotation exercises. (The patient is far above the average intelligence of the Navy's civil employees and likely to help.) September 16, 1920, he was "almost normal" as to motion and function.

Last seen October 20, 1920; he had practically recovered, without operation.

Case XII. J. P., aged thirty-four years. Shoe-hand. March, 1921, fell twelve feet, landing on heels. Examined by F. J. C., May 9, 1921. (Referred by the Federal Mutual Liability Insurance Company.) A small man; not robust; walks with slight limp. The limp, analyzed, is a failure of the clean "take-off" on the right foot. No flattening of "arch." A good deal of thickening below the external malleolus with limitation of pronation and supination reduced to about one-third normal.

Physiotherapy treatment recommended and now being carried out.

He is gaining satisfactorily and will probably be restored to an approximate normal grade without operation. Motion June 21, 1921, almost normal.

Case XIII. D. V. A case of avulsion fracture—operated with entire recovery—does not really belong in this series.

Case XIV. Lahey. Fresh fracture, reduced by mallet remodeling. Now, after six months, back on his job as apartment-house janitor. Not quite as good as new, but serviceable.

Case XV. H., machinist. Fresh fracture of os calcis from fall. Sent to my service at Public Health Nospital, No. 36, and reduced by the usual remodeling with the hammer and freeing of the joint motion with the hand. Seen June 7, 1921, three months later. Is back on the job, practically normal in stance and in motion.

Case XVI is one on my service at the City Hospital on which I have no record data at the moment. This was a boy of twelve with a cracked os calcis on one side, a fractured os calcis with displacement on the other—a fracture not only with displacement but with flattening and thickening as usual. Grace to his years, he has done well, and now, about eight months later, the case has become one of flaccid flat-foot on both sides, now being handled by plates and exercises, with notable good results so long as he wears the "rocker" plates. There is no disability with the pronation corrected.

Let us not forget that these heel-lesions carry a very large chance of flat-foot in convalescence even if there is no bony valgus deformity. Flat-foot symptoms in os calcis cases can be treated orthopedically with physiotherapy after care.

Cases with marked thickening to the outside, with pain on walking or standing, nearly always call for operative interference, soon or late; they can, however, be so handled as to do away with most of the deformity, with most of the loss of motion and with most of the symptoms. That this is possible is not sufficiently understood.

It is true that no one of the many crippled from injuries of this sort should be let drift along for many months without a trial of more modern methods. All of them, I think, can be improved by operation and most of them set in the way of an improvement that presently brings them back into the wage-earning class.

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3. Never published.

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5. Partly, at least, owing to a campaign of reform by the Accident Board. These are primarily painters' and roofers' accidents: the reform accomplished (through the bait of preferential insurance rates) has brought about inspection of ropes and pulleys, and a decrease in os calcis lesions.

6. For instance, I have found it practicable to remove nearly all of a comminuted scaphoid bone, then to setthe foot in proper shape and to secure a total replacement of missing bone.

7. I hold no brief against this tenotomy, and do occasionally do it. *Only*, I recognize the disadvantages, and do it only when I must.

8. In the evaluation of these results, please allow me a bit of leeway for personal judgment. I know that two of these men are not yet back at work, but jobs are scarce at present in Massachusetts, and our industrial compensation is pretty liberal.

9. Anterior arch troubles seem curiously frequent in these cases. They yield to usual handling. There is also, at times, a pain in the outer toes that is probably due not to statics but to interference with the nerve—also rather common, but fortunately temporary.

10. This was one of those common but yet distressing cases in which a year's idleness means forty pounds gain in weight, otherwise the interval might have been shorter.

11. This complaint is common, I find, in non-operative as well as operated cases, due, no doubt, to nerve pressure. In no case has it been of serious severity.

12. Operated on June 21st. Posterior-astragalo-calcaneal joint practically destroyed; restoration of lateral motion not very successful at operation, but return of function, including this motion, very satisfactory up to last visit, August 10th.

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